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CONGENITAL MALARIA

by

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Early Records

The question whether malaria can be transmitted from mother to child, either before or at the time of parturition, was a subject of discussion many years before the discovery of the malaria parasite. THOMAS of Leipzig (1886) cited cases recorded as examples of intra-uterine transmission by DINSTL, SCHRAMM, DUSCHEK and PLAYFAIR. BOHN (1873) referred to the same cases, and also to one reported by STEINER. It seems probable that two at least of these, those of Duschek and of Bohn, were instances of true congenital malaria, that is, malaria which the unborn child acquires from its mother owing to failure of the barrier action of the placenta *(BLACKLOCK and GORDON, 1925*a*). In Duschek's case, the infant, born of a malarious mother, died 3 hours after birth, and deposits of pigment were found at autopsy in the enlarged spleen and in the portal vein. Bohn's case was that of a premature dead infant, which also showed pathological evidence of malaria at autopsy. Leon HITTE (1902) wrote a thesis on the subject, citing two personal cases observed in Algiers, in

*Some writers (*e.g.* LAFFONT) confine the term congenital malaria to cases in which parasites are found in the blood of the child at birth, applying the term "hereditary malaria" to cases in which the infection is acquired from the mother, but where the parasite is not found until some days later.

each of which parasites were found in blood smears taken from the pulsating umbilical cord, and 19 from other sources. These records, along with a number of others reported by various observers, were subjected to critical analysis by BEEKEL (1910), who calculated that at least 11 of them were genuine. These included Hitte's two personal cases, two others cited by him (those of Duschek and Bohn), one reported by CRANDALL (1893) from New York and 6 recorded by ECONOMOS (1907), in each of which parasites were found in the peripheral blood of the child within 48 hours of birth. As the result of his analysis of all the evidence then available, Beekel concluded that transplacental transmission of malaria was possible, either by mingling of maternal and foetal blood at the moment of separation of the placenta (as suggested by PETERS, 1902), or by pathological or normal solution of the continuity of the placental syncytium. He considered, however, that such a mode of infection must be a rare event.

The existence of congenital malaria was generally accepted by early writers, who saw no reason to question it, but after the discovery of the malaria parasite by LAVERAN in 1880, a number of observers (MARCHIAFAVA, BASTIANELLI, BIGNAMI, CACCINI, THAYER, SCHAUDINN), who had made a special study of the disease, failed to find parasites in the foetal blood, umbilical cord or foetal surface of the placenta, in cases where the peripheral blood of the mother had been found positive, and concluded from this that intra-uterine transmission could not occur. PEZOPOULOS and CARDAMATIS (1907), as a result of their studies in Greece, took the same view, although in one of six cases two parasites were observed after prolonged search of smears taken from the foetal surface of the placenta.

For several years, doubts continued to be expressed in certain quarters, particularly in Italy, as to the possibility of transplacental transmission of malaria. As lately as 1911, SABELLA published an article in which, after citing 26 authorities, he recorded his conviction that congenital malaria did not exist (FORBES, 1923). Since that time, numerous papers dealing with the subject have appeared in the medical literature, and much confirmatory evidence as to the existence of true congenital malaria has been forthcoming. On a number of occasions plasmodia have been detected in smears from the umbilical cord, or in the peripheral blood of infants at birth, or within a space of time not exceeding the shortest pre-patent period of the species of parasite concerned.* In other instances, parasites have been demonstrated in the brain, liver, spleen or heart blood of the foetus (JEAN and VAN NITSEN, 1927; MACHWILADSE, 1928; DE FEYTER, 1932; WICKRAMASURIYA, 1935; GARCIA, 1938; GREEN, 1938; PERVES, 1943).

The literature relating to congenital malaria and to the effects of malarial infection on the course of pregnancy, on the foetus and on the newborn child falls into one of two categories. The first contains records of observations made on indigenous populations of malarious regions possessing a high degree of tolerance to malaria; the second consists of individual case reports, the majority of which refer to non-immune subjects temporarily resident in, or recently returned from, countries in which the disease is endemic.

Observations on Indigenous Populations of Endemic Areas

In an account of their investigations in Macedonia, PEZOPOULOS and CARDAMATIS (1907) drew attention to the massive infection that may be present in the placental blood at a time when only a few parasites can be seen in the peripheral blood of the mother. They also noted the preponderance of

*In a case reported by LUMBOS (1948), gametocytes of *P. falciparum* were found in the peripheral blood of an infant 22 hours after birth.

schizogony forms in the placental blood. These findings were confirmed and extended by CLARK (1915) from a study of 400 routine labour cases in Panama. Placental infection with *Plasmodium falciparum* was observed in 19 cases, distributed as in Table I.

TABLE I

Race	Number examined	Number with placental infection	Percentage with placental infection
North Americans (white)	118	0	0·0
Latin Americans (mixed Spanish and Red Indian)	92	3	3·26+
Europeans (white)	17	1	5·88+
West Indian Negroes	173	15	8·67+
Total	400	19	4·75

Of the 19 placental positive cases, parasites were found in the peripheral blood of the mother in 8 only, and in every instance where the peripheral blood of the mother was positive, the associated placental smear showed far more abundant evidence of infection. There was a great preponderance of schizogony forms in the placental blood, segmenting and pre-segmenting forms being the commonest types of parasite encountered. Crescents (gametocytes) were rarely seen, though these were common in smears made from the bone marrow and spleen. There was only one case classed as congenital malaria in the series, parasites being observed in a smear from the umbilical cord, and this was attributed to a placental accident. The author recommended the routine examination of the placental blood as the easiest and most certain method of diagnosing the presence of malarial infection in labour cases. He remarked on the peculiar suitability of the mature placenta as a harbour for the development of mature asexual forms of *P. falciparum*, by reason of its sluggish blood sinuses with their extensive epithelial lining, and its higher internal temperature. Mention of this last feature was prompted by certain (unpublished) observations of S. T. DARLING, who by puncture made immediately after death in a limited series of cases found a somewhat higher temperature in the uterus, spleen and liver than in the oral cavity, thorax and rectum (CLARK, personal communication, 1950). Later, in an investigation carried out in Panama in 1928, THONNARD-NEUMANN (1932) reported a placental infection rate of 20 per cent., associated with a maternal peripheral blood index of only 5 per cent.

BLACKLOCK and GORDON (1925*a* : 1925*b*) made an exhaustive study of the pathology of the placenta in malignant tertian malaria and of the relationship of malaria to labour and early infancy in Sierra Leone, West Africa. Parasites of *P. falciparum* were observed in the peripheral blood of only 9 out of 173 parturient women, pigmented leucocytes being noted in 3 others ; while out of 155 placentas in this series, 59 (38 per cent.) were found to harbour parasites of *P. falciparum*, with numerous schizogony forms. Crescents were never observed. Although the villous spaces of the placenta contained abundant parasites in the infected cases, in no instance were any observed in the vessels of the villi, in smears from the umbilical cord, or in the peripheral blood or organs of the infants. Among the many interesting features in this paper was a case of binovular twins, born with cords attached to different placentas. One of the placentas, together with the first 6 inches of the cord, was intensely

anaemic and contained numerous parasites, 30 per cent. of which were segmenting, with evidence of great destruction of erythrocytes. The child died within 15 hours. The other placenta and the whole of the cord attached to it were normal in appearance. There were fewer parasites in the placental blood, no segmenting forms and no evidence of destruction of erythrocytes. The child remained healthy during the 7 days of its stay in hospital. The authors suggested that the extreme destruction of erythrocytes seen in the first placenta might have been due to toxins liberated during segmentation.

Commenting on the fact that developing crescents were never found in the placenta, Blacklock and Gordon concluded that either there is some character in this organ which renders it unfavourable for the development of sexual forms of the parasite, or else the failure of crescents to develop is an indication of some degree of immunity. But they pointed out that if the latter explanation be accepted, it is difficult to imagine how in the same organ there can exist a degree of immunity sufficient to prevent the formation of crescents, and at the same time a lack of immunity which permits the development of asexual parasites to maturity and segmentation in enormous numbers.

TABLE II

Records of Congenital Malaria among Indigenous Inhabitants of Highly Malarious Areas

Author	Locality	Number of infants	Percentage of infants infected	Percentage of placentas infected	Percentage of mothers infected
CLARK (1915) ...	Panama ...	400	0·25	4·75	2·0
BLACKLOCK & GORDON (1925*b*)	Freetown, Sierra Leone ...	173	0	38	5·2
VAN DEN BRANDEN (1927)	Leopoldville, Belgian Congo	55	0	1·8	56
LOMBART (1931) ...	Elizabethville, Belgian Congo	50	2·0	50	56
SCHWETZ & PEEL (1934)	Stanleyville, Belgian Congo	56	3·6	74	68
PEEL & VAN HOOF (1948)	Leopoldville, Belgian Congo	393	0	49	65
GARNHAM (1938) ...	Kisumu, Kenya	404	0	27	30
GARNHAM (1949) ...	Kisumu, Kenya	146	0*		
BUTLER (1930) ...	Gold Coast ...	328	†	23	18
WALTON (1947) ...	Freetown, Sierra Leone	2,154	0·32	4·8	5·5
BRUCE-CHWATT (1950)	Lagos, Nigeria ...	543	0·18	19	10
PERVES (1943) ...	Abong M'Bang, French Cameroons	661	9·6		44
DALÉAS & LAVERGNE (1936)	Indo-China ...	887	0·3		1·1

*There was one possible case of congenital malaria (*P. malariae*) in this series, but the author considered that the infection was probably acquired during parturition. Mosquito transmission could be almost certainly excluded, as the event occurred in the "off-season" and the child lived in a protected part of the town.

†The blood of 50 children in this series was examined, and malaria parasites were found in one instance.

Since the publication of Blacklock and Gordon's paper, a number of other observers have undertaken investigations on similar lines. The results of these are summarized in Table II. In the majority of cases the extreme rarity of congenital malaria among the indigenous populations of highly malarious areas has been confirmed : in the Belgian Congo by VAN DEN BRANDEN (1927), LOMBART (1931), SCHWETZ and PEEL (1934), PEEL and VAN HOOF (1948) ; in Kenya by GARNHAM (1938 : 1949) ; in Sierra Leone by WALTON (1947 : 1949) ; in the Gold Coast by BUTLER (1930) ; in Lagos by BRUCE-CHWATT (1950). In the French Cameroons, however, PERVES (1943 : 1946) recorded malarial infections in 9·6 per cent. of newborn infants, associated with a maternal parasite rate of 44 per cent.

Outside Africa, the only extensive series of observations among indigenous populations of malarious areas on record is that of DALÉAS and LAVERGNE (1936), who reported infections of smears from the umbilical cord in 887 labour cases in Indo-China. Henry's melano-flocculation test was, however, found positive in 28 of the infants, and the authors concluded on this score that congenital malaria was a not uncommon event in Indo-China, despite the rarity with which parasites have been observed in the peripheral blood. ENGLISH (1946) stated that in a maternity hospital in Singapore in 1938 there were 241 cases of malaria in mothers, but that in only one instance (a European) were parasites found in the blood of a newborn infant.

There is some evidence that congenital malaria is not infrequent in certain parts of Eastern Europe. A report on the visit of the League of Nations Malaria Commission to that region (SWELLENGREBEL, 1925) contains a reference to a series of 40 cases recorded in Skradin, Dalmatia and of 8 cases in Persianovka, Don, in all of which parasites were found in infants within 5 days of birth. WESELKO (1926) in Albania cited 14 cases in which parasites were found in the peripheral blood of both mother and child immediately after birth or within 5 days thereafter. LJACHOWETSKY (1927) reported several hundred cases diagnosed as congenital malaria from the Crimea, though in many of these parasites were not seen early enough to exclude mosquito transmission with certainty. LOPATIN (1940) reported 15 *vivax*, 10 *falciparum* and 8 *malariae* cases from Kuibyshev, in the Middle Volga area of South Russia, " occurring within 14 days of birth ". In none of these reports from Eastern Europe, however, is the percentage stated of congenital malaria to total births.

Data regarding infection of placental blood among indigenous populations were recorded by VAN DEN BRANDEN (1927), BUTLER (1930), LOMBART (1931), SCHWETZ and PEEL (1934), GARNHAM (1938), PEEL and VAN HOOF (1948), WALTON (1949) and BRUCE-CHWATT (1950). The findings of PEZOPOULOS and CARDAMATIS, CLARK, and BLACKLOCK and GORDON in this respect were substantially confirmed in all cases with one exception, that of van den Branden, who found only one infection (with *P. malariae*) out of 55 placentas examined at Leopoldville, Belgian Congo, although parasites were observed in the peripheral blood of 31 mothers in the same series.* This finding is in striking contrast with other reports emanating from that country, where Lombart found 50 per cent. of placentas infected, Schwetz and Peel 74 per cent., and Peel and van Hoof 49 per cent. In 6 out of 14 cases cited by WESELKO (1926) in Albania, parasites were found in the placental blood.

Records of Individual Cases diagnosed as Congenital Malaria

A summary of 28 cases reported from various sources was given by FORBES (1923) and is reproduced in Table III. Of the 13 cases in which the species of

*According to DUBOIS (discussion on PEEL & van HOOF, 1948) there is some doubt as to whether the smears examined by van den Branden were taken from the maternal or foetal side of the placenta.

parasite is mentioned, 8 were *P. vivax* infections, 3 *P. falciparum* and 2 *P. malariae.*

TABLE III

Summary of 28 Cases Diagnosed as Congenital Malaria [after Forbes, 1923]

Author	Number of Cases	Age malaria discovered in infant	Plasmodia in infant's blood	Plasmodia in mother's blood	Remarks
BOHN (1873)...	1	Premature, dead			Necropsy evidence
MEARA (1909)	1	7 weeks ...	Yes ...	Yes ...	
DUSCHEK (quoted by BOHN)	1	3 hours ...			Necropsy evidence
CRANDALL (1893)	1	72 hours ...	Yes ...		
ECONOMOS (1907)	6	Within 48 hours	Yes ...		
HITTE (1902)	2	At birth ...	Yes ...		Blood from cord
BEEKEL (1910)	1	7 weeks ...	Yes (Q)	No ...	
BASS (1914) ...	2	7½ weeks...	Yes (BT)	No ...	
		At birth ...		Yes ...	
THIBAULT(1915)	4	3 hours ...	Yes (BT)		
		2 days ...	Yes (BT)	Yes (BT)	
		5 days ...	Yes (MT)	Yes (BT)	
		Stillbirth...	Yes (MT)	Yes (BT)	
JONES, D.W. & MOORE, H.L. (1918)	1	9 days ...	Yes ...		
RABINOFF (1920)	1	Few hours	Yes (BT)		
LEMAIRE, DUMOLARD & LAFFONT (1910)	1	At birth ...	Yes (Q)	Yes (Q)	No symptoms till 40 days old
TISSIER & BRUMPT (1913)	1	19 days ...	Yes (BT)	Yes (BT)	Born in Paris
LEGER (1918)	1	Stillbirth (7 months)	Yes (BT)	Yes (BT)	
HEISER (1913)	1	5 days ...	Yes (MT)		
BLOOM (1918)	1	3 weeks ...	Yes (BT)	Yes (BT)	
CUADRA (1920)	1	2 days ...	Yes (BT)	Yes (BT)	
PENNATO (1921)	1	Stillbirth...			Necropsy evidence

Data relating to 107 other cases are summarized in Table IV. No case has been included unless the parasite was observed in the infant within 7 days of birth, or unless by reason of locality or season mosquito transmission can be excluded. In 34 of these cases the species of parasite recorded was *P. vivax*, in 43 cases *P. falciparum* and in 2 cases *P. malariae.* There are 25 others, 20 from the Belgian Congo and 5 from northern India, in which the parasite was almost certainly *P. falciparum.*

TABLE IV

Summary of 107 Cases Diagnosed as Congenital Malaria

Author & Locality	Species of Parasite found in			Age of Infant	Remarks
	Mother	Placenta	Infant		
ALESSANDRINI (1931), Italy	MT	MT	MT	At birth	
ANNECCHINO (1926), Italy	BT		BT	4 days...	Schizonts and gametocytes in blood of both mother and child
ARIAS ARANDA (1926), S. America			BT	2 days...	Schizonts and gametocytes. Child died 2nd day
BOURGIN (1935), Indo-China	BT		BT	At birth	Gametocytes in blood of both mother and child
BRINDEAU (1926), Paris	BT		BT	4 weeks	Occurred in mid-winter. Mother infected in Morocco
BUCKINGHAM (1925), China	?		?	At birth	Parasites found before expulsion of placenta
			Q	Stillbirth	Parasites found in smear from prolapsed hand before birth
DE CAPRIO (1944–45), Italy	BT		BT	At birth	Both parents and child infected with syphilis
CHORINE (1932), Indo-China	BT		BT	At birth	
CHUNG *et al.* (1948), China	BT		BT	7 days...	
COLUZZI (1948), Italy	BT		BT	2 days...	
	BT		BT	2 days...	
	BT		BT	4 days...	
	BT		BT	5 days...	
	BT		BT	7 days...	
DAS GUPTA (1939), India	BT		BT	15 hours	Mother had repeated attacks during pregnancy
DOROLLE & DANG-HUU-CHI (1927), Indo-China	MT		MT		Mother died from malaria when 5 months pregnant
DUMOLARD & VIALLET (1909), Algeria	BT		BT	1 hour...	Child died 1 hour after birth. Parasites in cord and heart blood
ENGLISH (1946), Singapore	MT		MT	At birth	
DE FEYTER (1932), Congo	MT		MT	At birth	
	MT		MT	7 days...	
	MT		MT	Stillbirth	Six months' foetus. Parasites in cord blood and in spleen

TABLE IV (*continued*)

Author & Locality	Species of Parasite found in			Age of Infant	Remarks
	Mother	Placenta	Infant		
FORBES (1923), Colorado, U.S.A.	Q		Q	7 weeks	Mother acquired infection in Greece
GAMMIE (1944), England	BT		BT	4 weeks	Occurred mid-winter. Mother acquired infection in India
GARCIA (1938), Philippines	MT	MT	MT		Mother died at 6th month. Parasites in cord
	MT		MT		Mother died at 4th month. Parasites in heart blood of foetus
GREEN (1938), W. Africa	MT		MT	2 days ...	Parasites in vessels of child's brain
	MT		MT	At birth	
	MT		MT	2 days ...	
HOUEL (in LAFFONT & FULCONIS, 1931), Algeria	BT		BT	18 days ...	Locality free from *Anopheles*
JAGDSCHAN (1924), Russia		MT	MT	At birth	Crescents in child's blood
JAHIER (in LAFFONT & FULCONIS, 1931)	BT		BT		
JEAN & VAN NITSEN (1927), Congo			*	Stillbirth	Six months. Schizonts in spleen
			*	Stillbirth	Six months. Schizonts in spleen
			*	Stillbirth	Six months. Schizonts in spleen
			*	Stillbirth	Five months. Schizonts in spleen
			*	Stillbirth	Eight months. Schizonts in spleen
			*	Stillbirth	Nine months. Schizonts in spleen
			*	1 day ...	
			*	2 days ...	
			*	2 days ...	
			*	2 days ...	
			*	2 days ...	
			*	2 days ...	
			*	2 days ...	
			*	2 days ...	
			*	5 days ...	
			*	5 days ...	
			*	5 days ...	
			*	5 days ...	
			*	5 days ...	
JONES (1950), W. Africa			MT	At birth	Child died 2nd day
	MT		MT	At birth	Child died 4th day
			MT	At birth	Child died 4th day
JONES & BROWN (1924), England	BT		BT	16 days ...	Gametocytes in child's blood

TABLE IV (*continued*)

Author & Locality	Species of Parasite found in			Age of Infant	Remarks
	Mother	Placenta	Infant		
LADYJENSKI (1930), Russia	MT		MT	At birth	
	MT	MT	MT	At birth	
	MT	MT	MT	At birth	
	MT	MT	MT	At birth	
	BT	BT	BT	At birth	
	BT	BT	BT	At birth	
LAFFONT, BINET & LESINI (1929), Algeria	BT		BT	At birth	Child died 12th day
LAFFONT & FULCONIS (1931), Algeria	BT		BT	At birth	Child died 28th day. BT schizonts in spleen
VAN LAWICK VAN PABST (1949), Java	MT		MT	At birth	
	BT		BT	At birth	Six months' child died after 1 hour
LIEM TJAIJ TIE (1941), Indonesia			BT	At birth	
			MT	2 days ...	
LIEVRE (1934), Algeria	MT		MT	At birth	
LIMBOS (1948) ...	MT		MT	22 hours	Crescents in child's blood
LIPSCOMB & MANSELL (1927), India			BT	26 days ...	Child born at Murree, 7,200 ft. No local mosquito transmission
LYNCH (in HANSON, 1928), U.S.A.			?	Within 3 days	Species of parasite not mentioned
			?	Within 3 days	
MACHWILADSE (1928), Russia	MT, BT, Q		MT	At birth	Child died after 1 hour. Parasites in liver smears
MACKAY (1933) Tanganyika			MT	At birth	Parasites in smears from child's heel
			MT		Parasites in smears from child's heel
			MT		Parasites in smears from child's heel
			MT		Parasites in smears from child's heel
			MT		Parasites in smears from child's heel
			MT		Parasites in smears from child's heel
	MT		MT		Parasites in smears from child's heel
MEASHAM (1938), India	BT		BT	17 days ...	Born in non-transmission season
MOFFAT (in JEAN, 1927), England			MT	7 weeks	Mother returned from Africa 5 months before delivery
VAN NITSEN (1934), Congo	MT		MT	2 days ...	
			MT	3 days ...	

TABLE IV (*continued*)

Author & Locality	Species of Parasite found in			Age of Infant	Remarks
	Mother	Placenta	Infant		
PIK GING HOO (1941), Amsterdam	BT		BT	20 days ...	Gametocytes in child's blood. Chance of mosquito transmission remote
PINELLI (1932), Italy	BT		BT	5 days ...	Gametocytes in child's blood
RODHAIN (1933), France	BT		BT	2 months	Mother infected in Congo
SCHADOW (1931), Germany	BT		BT	3 weeks	Mother treated with malaria therapy during pregnancy
SOEDARSONO (1941), Indonesia	BT		BT	1 day ...	
	BT		BT	8 days ...	Child died 8th day. Mature parasites in blood
STRICKLAND & BAIRD (1939)			*	16 hours	
			*	3 days ...	
			*	5 days ...	
			*	7 days ...	
			*	7 days ...	
TANNER & HEWLETT (1935), England			BT	53 days ...	One of twins born in London. Mother acquired infection in India
THIODET (in LAFFONT & FULCONIS, 1931), Algeria	BT		BT	2 days ...	
THONNARD-NEUMANN (1931), Columbia	MT		MT	At birth	Mother syphilitic
VALCKE (1925), Congo			*	2 days ...	
VALCKE (1927) ...	MT		MT	1 day ...	Mother had blackwater fever
WAYL (1934), Egypt	MT		MT	6 days ...	
	MT		MT	3 days ...	
WICKRAMASURIYA (1937), Ceylon ...	MT	MT	MT	At birth	Mother died from hyperpyrexia
	MT	MT	MT	Stillbirth	Parasites in foetal brain and spleen. Mother died
	MT		MT	Stillbirth	Parasites in foetal spleen, liver, heart. Mother died
		MT	MT	Stillbirth	Parasites in foetal brain and spleen. Mother recovered
	MT		MT	Undelivered	Mother died in coma

*All these cases were probably MT infections.

Two of the quartan cases, those reported by Beekel (Table III) and by Forbes (Table IV), are of particular interest. Both cases occurred in the U.S.A. and in both the parents were immigrants from malarious European countries. In Beekel's case the parents were Hungarian, and the mother had suffered from malaria in adolescence, but had experienced no overt attack for several years. The child was born in Cleveland, Ohio, and was brought to hospital at the age of 10 weeks, with a history of 3 weeks' fever. The child was extremely anaemic, the spleen was hard and projected 3 cm. below the costal margin and the liver was also enlarged. Quartan parasites were present in large numbers in the peripheral blood, including pre-segmenting and segmenting forms. The spleen of the mother was palpable, but repeated examinations of the peripheral blood proved negative. The child had been born on September 28, after which time mosquitoes are uncommon in the locality, which is in any case regarded as being non-malarious. Mosquito transmission of so rare an infection as quartan malaria under such circumstances is extremely unlikely. In the case reported by Forbes, the parents were Greek, but had resided in the U.S.A. for 5 years preceding the birth of the child. The mother had suffered from quartan malaria in Greece, but had had no overt attack for $4\frac{3}{4}$ years. The child, who was born in Colorado, developed fever at the age of 7 weeks, and had 6 febrile paroxysms at intervals of 72 hours before the significance of the periodicity was fully appreciated. At the 7th febrile attack, the child then being 10 weeks old, the spleen was enlarged, the child anaemic, and quartan parasites in all stages of the asexual cycle were numerous in the peripheral blood. The mother's blood was also found to contain quartan parasites at this period, although the spleen was not palpable and there was no fever. Mosquito transmission in this case could be excluded with certainty, since there are no *Anopheles* mosquitoes in eastern Colorado (Cockerell, 1918) from where this record came.

Another case of interest is that reported by Tanner and Hewlett (1935). The mother, an Englishwoman married to a Bengali, who had suffered from repeated malarial attacks during pregnancy, returned to England from India in September 1934. In the following January she gave birth in London to binovular twins, the second of whom was admitted to hospital 50 days later with a spleen projecting 4 cm. below the costal margin, a red cell count of 2,310,000 per cmm. and benign tertian parasites in the peripheral blood. The other twin remained healthy.

Records of examination of placental blood are given in only very few of these individual case reports. Out of all those listed in Table IV, the placental blood was apparently examined in 10 only. It is presumably this omission to examine smears from the placenta in the majority of cases which has given rise to an impression in some quarters that placental infection in non-immune subjects is extremely rare (Jones, 1950). Such evidence as is available lends no support to this impression. For example, Yorke and Murgatroyd (1931) reported the case of a European woman infected in West Africa with *P. falciparum* who gave birth to a child in England. Section of the placenta showed a massive infection, though parasites were not observed in the blood of the baby. Wislocki (1930) also recorded heavy infection of the placenta in 4 out of 6 labour cases studied during the great Ceylon malaria epidemic of 1934–35, when the general level of immunity among the local inhabitants was extremely low. Three of the 4 mothers actually died from malaria in labour or within a few hours after parturition.

The Mechanism of Transplacental Transmission of Malaria

Although the fact that congenital malaria can occur is no longer in dispute, the manner in which the infection is transmitted from mother to foetus is as much a matter of conjecture as was the case 40 years ago. There is general

agreement that in certain cases there is a breakdown in the protective barrier of the placenta which allows the parasite to pass, but there is considerable divergence of opinion as to how this comes about.

When the developing ovum reaches the uterus, it becomes embedded in the thickened mucous membrane (decidua), to which it attaches itself by means of its outer layer or chorion, processes of which penetrate into the decidua. The chorion and its processes are covered by a thick syncytium termed the trophoblast ; this burrows its way into the uterine mucous membrane and gives off villus-like chorionic processes (chorionic villi) which enter the large vascular sinuses in the decidua, where they become bathed with arterial maternal blood. In the meantime, tissue conveying blood vessels has grown into the chorionic villi from the mesoderm of the foetus, bringing to them foetal blood by way of the umbilical arteries. In the later stages of pregnancy, the original epithelial covering of the villi becomes attenuated, and only a thin syncytial layer of cells separates the tissue of the villus containing foetal capillaries from the maternal blood in the sinuses. After the birth of the child, a separation takes place in the substance of the decidua and the placenta becomes detached from the uterus. During this process the uterine blood vessels are necessarily ruptured, but the orifices of the torn vessels are closed by the firm contraction of the uterine muscular fibres, and thus post-partum haemorrhage is prevented.

In the course of a discussion on a paper by Garnham (1938), read before the Royal Society of Tropical Medicine and Hygiene in London, Green-Armytage remarked that there seemed to be no real anatomical reason why congenital malaria should not be extremely common, not only because of the delicacy of the degenerating syncytial membrane in the later stages of pregnancy, but also because of the risk of trauma (whether incidental or resulting from toxaemia and accidental haemorrhage) breaking up the continuity of the ectodermal covering. He added that in his own extensive obstetrical practice in India he had only seen one case. English (1946) also expressed surprise that there were not more cases of congenital malaria on record, considering the intimacy of the maternal and foetal blood streams.

Despite the apparent vulnerability of the placental barrier, however, the fact that massive *falciparum* infections can exist in that organ in association with complete absence of parasites in the blood of the umbilical cord and of the foetus, shows that the walls of the villi are normally a very effective safeguard. Pennato (1921) noted the protective action of the placenta against certain poisons, as instanced in a case of fatal mercury poisoning in a mother, the infant remaining unaffected. This author also drew attention to the fact that placental transmission of syphilis does not take place if the infection is contracted by the mother in the last two months of pregnancy. That the placenta possesses certain selective powers is evidenced by the fact that glucose is more plentiful in the maternal than in the foetal blood ; while the proportion of iron, lime and potash is greater in the foetus than in the mother during the last months of pregnancy.

It has been assumed that leucocytes can pass from the maternal to the foetal circulation, on the ground that the blood in the umbilical vein contains per volume a greater number of white cells than that in the umbilical artery. Commenting on this assumption, Blacklock and Gordon (1925*b*) noted that while leucocytes containing malaria parasites are not infrequently present in the placental blood and leucocytes containing malarial pigment are commonly seen therein, neither of these were ever observed by them in the circulation of the newborn infant. Moreover, even if leucocytes can pass the placental barrier, it does not follow that erythrocytes can do so, still less those infected with parasites, since it was shown by Marchiafava and Bignami (1894) that in capillary apoplexies almost all erythrocytes are without parasites, even

when infected cells are present in large numbers in the cerebral vessels. However, it seems in any case unlikely that leucocytes can pass the placental barrier, for in leukaemia these may be present in the maternal blood in enormous numbers, without a corresponding increase in that of the foetus. The efficacy of the placenta as a barrier against the entry of leukaemic cells into the foetal circulation was remarked upon by CAMERON (1888–1890).

In view of the undoubted efficacy of the placenta as a protective barrier and the relative rarity of congenital malaria even in cases of massive placental infection, many observers have assumed that mechanical damage to the placenta is an essential precursor of malarial transmission (*e.g.*, CONCETTI, 1912 ; CLARK, 1915 ; DOROLLE and DANG-HUU-CHI, 1927 ; JEAN, 1927 ; THOMSON and ROBERTSON, 1929 ; LADYJENSKI, 1930 ; THONNARD-NEUMANN, 1932 ; ALESSANDRINI, 1931 ; VAN NITSEN, 1932). The view most frequently expressed by those who hold this belief is that whilst the placental filter normally prevents the passage of the malaria parasite, small infarcts or capillary lesions may result in this obstacle being overcome.

WICKRAMASURIYA (1937), as the result of his studies in Ceylon, challenged the validity of this theory. In none of the 5 cases of undoubted congenital malaria reported by him was there any sign of macroscopical damage to the placenta, and he therefore concludes that the lesion must have been of a different nature. He contended that the massive infection and the high temperature engendered by such an infection, together with the effects of the toxic products of the parasites, must induce pathological changes in the placental substance, and thought it probable that a breach in the protective barrier might be effected as the result of such pathological changes. He admitted the likelihood that if labour supervened the uterine contractions might force more and more parasites into the foetal circulation, once a breach had been created in the placenta, but since in two of his cases mother and foetus had died before the onset of labour, this could not be an essential factor in transmission. The view that malarial infection in the newborn indicates pathological changes in the placenta, thus facilitating the passage of parasites, was also put forward by PIK GING HOO (1941).

VAN LAWICK VAN PABST (1949) reported two cases of congenital malaria in his own practice in Java in which there was no macroscopical damage to the placenta and no sign of infarcts or haemorrhages. He disagreed with the view that such lesions are causative factors in the transplacental transmission of malaria, on the ground that in such cases fibrin is formed around the trophoblast of the villi, cutting off nourishment and causing their obliteration, a process which does not bring about an open circulation between the maternal and foetal blood. He offered two alternative suggestions : (i) that exo-erythrocytic forms of the parasite may penetrate the villi, or (ii) that sporozoites may do so.

LAFFONT and FULCONIS (1931) cited two cases of congenital malaria occurring in Algiers in which there was no evidence, either macroscopic or histological, of any damage to the placenta. From a survey of all the cases recorded in Algeria up to that time, they concluded that transplacental transmission was dependent neither on intensity of infection, nor on the mother's physical condition, nor on the occurrence of recent infection. BUCKINGHAM (1925) also drew attention to the absence of infarcts or any evidence of premature separation or other apparent damage to the placenta in two cases of congenital malaria which occurred in his practice in China.

JEAN and VAN NITSEN (1927) recorded a series of 34 cases in which 18 newborn infants were found to harbour malaria parasites, while in the same series microfilariae were observed in the peripheral blood of 14 of the mothers. No microfilariae were seen in any of the infants. They concluded from this that it was not by placental haemorrhages that foetal malarial infection

occurred. The presence of microfilariae in large numbers in the peripheral and placental blood of the mother, but not in the foetal circulation, was also noted by GARNHAM (1938).

Some observers, *e.g.*, SCHWETZ (1939), have advanced the view that febrile malaria during pregnancy is essential to permit the passage of parasites, by causing a greater friability and permeability of the placenta. There are, however, a number of instances on record which show that transmission can occur in the absence of active malaria, for example the cases of quartan infection cited by BEEKEL (1910) and by FORBES (1923) in America, and the 3 cases of malignant tertian infection reported by JONES (1950) in West Africa.

This point was discussed by DAS GUPTA (1939), who infected a pregnant *rhesus* monkey with *P. knowlesi*. The monkey was chloroformed when already moribund as the result of the infection, and the placenta was found to be packed with parasites. There was no evidence of malaria in the foetus, nor any sign of anaemia, in marked contrast to the condition of the mother. The same author reported a case of human congenital malaria (*P. vivax*) where the mother had had repeated attacks of malaria throughout her pregnancy, and concluded that it was the chronicity of the infection rather than the intensity which determined the passage of parasites.

ASCIONE and MARIOTTI (1932) claimed to have produced malarial infection in 10 subjects by injecting blood (5 cases) and cerebrospinal fluid (5 cases) from patients suffering from malaria, which had been passed through Berkefeld V and N filters, and suggested that the merozoites might go through a " filterable stage " and subsequently regenerate. TANNER and HEWLETT (1935), reporting on a case of congenital malaria, the second of binovular twins, referred to above, remarked that if this were true the parasites might pass the chrorionic syncytium in such a stage. They added, however, that such a mode of infection was disproved in their own case, since only one of the twins was affected, and suggested premature placental separation as the most probable cause of transmission. Subsequent observers have failed to confirm the observations of ASCIONE and MARIOTTI (PANAGIA, 1933 ; ZOZAYA and ESPINEL SABOGAL, 1933 ; MARCHOUX, LEROY and CHORINE, 1933 ; TROISIER and MODALDI, 1933).

From a study of all the available data, it is clear that none of the theories advanced to account for the transmission of malaria from mother to child is supported by concrete evidence. Congenital malaria can certainly occur in the absence of any demonstrable damage to the placenta, and in the absence of any sign of active malaria in the mother during pregnancy, and there is no evidence to support the suggestion that the parasite may pass the placental barrier in some exo-erythrocytic phase. The most likely explanation seems to be that transmission is rendered possible by some pathological damage to the placenta due to malaria, but the manner in which this is brought about and the reason why it should occur in some cases and not in others remain matters of speculation.

Frequency of Incidence of Congenital Malaria

Statistics regarding the incidence of congenital malaria are apt to be misleading. Some authors give percentages based on large series of unselected labour cases, and this may give rise to a totally wrong impression, since many of the mothers may not have been exposed to malaria infection during pregnancy. In other instances the figures given relate only to mothers in whose blood parasites have been found during the pregnancy, or to those who have actually experienced an overt attack of malaria during that period.

Most of the investigations carried out among indigenous populations of malarious countries where transmission occurs throughout the greater part of the year and infection is universal, indicate that under such conditions congenital malaria is exceedingly rare. Thus, CLARK (1915) in Panama recorded 1 case in 400 births, BLACKLOCK and GORDON (1925*b*) in Sierra Leone none in 173, WALTON (1949) in Sierra Leone 7 in 2,154, BUTLER (1930) in the Gold Coast 1 in 50, BRUCE-CHWATT (1950) in Nigeria 1 in 543, GARNHAM (1938) in Kenya none in 404 and in a second series one doubtful case in 149 (GARNHAM, 1949), VAN DEN BRANDEN (1927) in the Congo none in 55, LOMBART (1931) in the Congo 1 in 50, SCHWETZ and PEEL (1934) in the Congo 2 in 56, PEEL and VAN HOOF (1948) in the Congo none in 393 and DALÉAS and LAVERGNE (1936) in Indo-China 3 in 887. These records represent a total of 5,324 births with 16 congenital infections (0·03 per cent.). In the Cameroons, however, PERVES (1946) has recorded 63 cases of congenital malaria among 661 newborn infants, a rate of 9·6 per cent.

There are two reports, each relating to a short series of cases in hyperendemic areas of Africa which call for comment. MACKAY (1933) recorded 7 cases in which parasites were said to have been found in the peripheral blood of newborn infants in a series of 22 confinements at Dar-es-Salaam, Tanganyika ; whilst JEAN and VAN NITSEN (1927) in the Belgian Congo recorded 14 cases out of 34 in which parasites were observed in the peripheral blood of infants within 5 days of birth and 4 others in the same series from the 8th to the 12th day. In both these series the mothers were presumably Africans. It is impossible to reconcile these figures with others emanating from the same territories, and it is obvious that they cannot be accepted as valid without corroboration.

In a paper read at the Second International Congress on Malaria held in Algiers, LAFFONT and FULCONIS (1931) estimated the incidence of congenital malaria in the local indigenous population at approximately 4 per cent. In the discussion which followed, Rodhain remarked that this was a much greater frequency than had been observed in the Belgian Congo up to that time. LHUERRE (1928) considered that congenital malaria was common among the indigenous population around Dakar, though frequently overlooked. Similar statements have been made in regard to various countries in Eastern Europe, as already noted. It is noteworthy, however, that none of the statements alleging that congenital malaria is relatively frequent among indigenous populations of malarious countries is supported by figures except that of Perves in the Cameroons.

There is evidence that congenital malaria is probably more frequent among non-immune subjects than among those with a high degree of tolerance to the disease. THIBAULT recorded 3 instances out of 246 confinements occurring in Arkansas, U.S.A., over a period of 12 years, in which there was active malaria in the mother during pregnancy. VAN LAWICK VAN PABST (1949) reported an incidence of 2 per cent., in his own practice in Java (90 cases), JONES (1950) recorded 4 infections of the umbilical cord (all *P. falciparum*) out of 54 successive deliveries in Lagos, and concluded that among non-indigenous European women of relatively low immunity, infected with the local strain of *P. falciparum*, congenital transmission of malaria was by no means rare.

A number of theories have been advanced from time to time to account for the fact that congenital malaria is not more frequent than is actually the case. Thus PEEL and VAN HOOF suggested that the extraordinary number of phagocytes in the placenta may contribute an obstacle to the passage of parasites. GARNHAM suggested that except where there is gross placental damage, the characteristic "stickiness" of erythrocytes containing mature asexual forms of *P. falciparum* may inhibit their passage through the intervillous barrier. It cannot be said that any of the explanations put forward is entirely adequate.

Species of Parasite associated with Congenital Malaria

As the records show, congenital malaria can occur in infections with all three of the common species of human malaria parasite. Comparatively few quartan cases have been reported, but no fewer than may reasonably be attributed to the relative rarity of this species of parasite. Of the cases referred to in this review, excluding those in which the species of *Plasmodium* is not definitely stated, 57 were recorded as *vivax* infections, 56 as *falciparum* and 12 as *malariae*. Most of the cases reported in Europe and in North Africa have been due to *P. vivax*, but there have been a number of *falciparum* cases relating to European women temporarily resident in the tropics. It seems probable that the species incidence of congenital malaria depends on the relative local frequency of the different species of *Plasmodium*.

Transplacental Transmission of Malarial Immunity

There is a considerable body of evidence indicating that some degree of temporary immunity may be transmitted from mother to child. Clark (1915) noted that it was extremely rare at Ancon, Panama, to see at the morgue children under one or two years of age who had died of malaria. In a later paper (Clark, 1937), the same author remarked that in Panama infants enjoy almost complete protection from malaria in the first two months of life by reason of some quality inherited from the mother or associated with the nursing period. No babies were found with parasites in their blood before the third month, and the normal parasite index was not reached even at the age of one year. Blacklock and Gordon (1925*b*) presented a graph relating to the blood examination of 800 children in Sierra Leone of ages up to 2½ years. Only one positive case was recorded during the first month of life, while from then onwards the infection rate showed a progressive rise. The authors concluded that either the child at birth was endowed with a passive immunity, derived from the mother, which steadily diminished till the age of 1½ years, or that effective exposure to infection increases steadily as the child grows older till it reaches this age ; this second explanation seemed to them the less probable.

Barber and Olinger (1931) in Southern Nigeria recorded a parasite rate of 45 per cent. in 281 infants 3 to 4 months old in a locality where the sporozoite rate in *Anopheles gambiae*, the chief local vector, was 11 per cent. and the density of this species of *Anopheles* 8·6 per house. They argued that if very young infants enjoy no natural protection, it is difficult to imagine how a single one could escape infection even for 3 weeks, still less for 3 months. Later, in Macedonia, Barber and his colleagues in that country compared the parasite rate in infants of 2 to 3 months with that at 5 to 6 months in two series of cases, and in each instance found a significant increase in the older category. They inferred that some inhibiting substance might be transferred from mother to infant before birth or during lactation (Barber, Mandekos & Rice, 1936 : 1937).

Bagster Wilson (1936) in Tanganyika noted that almost all the blood examinations of infants in the coastal area around Tanga during the first month of life were negative. The general condition of the babies at this time was excellent ; they were well covered, placid and generally in better health than their mothers. A month or two later the picture was very different, the babies then being obviously ill, feverish, snuffling and constipated.

Garnham (1949) in Kenya concluded that the comparatively low parasite rate (10 per cent.) observed during the first 3 months of life was probably due to the presence of transmitted passive immunity. Bruce-Chwatt (1950) in Nigeria reached a similar conclusion, his figures for the blood examination of infants aged 3 months or under closely approximating those of Garnham.

Strickland, Sen Gupta and Mazumdar (1939) in a highly endemic area of northern Bengal, calculated that the average age of infants undergoing their first attack of malaria was 3 to 4 months. Coluzzi (1948), reporting 5 cases of congenital malaria (all *P. vivax* infections), noted that all the infants were readily cured with very small doses of quinine and that there were no relapses ; from which he inferred that the cures were probably spontaneous by reason of immunity acquired during gestation.

Other writers who have expressed similar views on the subject of intra-uterine transmission of immunity are Hackett (1941), Butler (1930) and Walton (1947).

Effect of Malaria on Pregnancy and on the Newborn Child

That malaria is responsible for a large number of interrupted pregnancies has long been recognized. Scheube (1903) stated that miscarriages were more frequent in European women in the tropics than in temperate climates, and attributed this chiefly to the influence of malaria. Orgeas (1883) expressed similar views with respect to European women in French Guiana. The harmful effect of malaria on pregnancy has been emphasized by Conti (1910) in Sardinia, di Pace (1924) in Italy, Laffont and Jahier (1931) in Algeria, Wise (1920) in British Guiana, Clark (1915) in Panama, Nagger (quoted by Anderson, 1927) in Egypt and Thomson (1935) in Nyasaland. Sinton (1935) cited a number of authors who have testified to the same effect in India (Robertson, 1909 ; Christophers, 1911 ; Bentley, 1911 ; Dyson, 1911 ; Newell, 1913 ; Das, 1923 ; Brahmachari, 1923 ; Watson, 1924 ; Gill, 1928). Christophers remarked on the great increase in the number of abortions, premature labours and stillbirths during the great Punjab epidemic of 1908. In Amritsar, in the autumn of that year, the number of still births recorded was 5 times the normal. It was estimated that in 4,600 pregnant women in that city, malaria caused some 300 premature births and 1,100 stillbirths, miscarriages and abortions, that is, 30 per cent. of interrupted pregnancies.

Wickramasuriya (1937), from his experience in the great Ceylon epidemic of 1934–35, concluded that malaria *per se* was a far more potent abortifacient than any drug, especially in ill-nourished persons suffering from ankylostomiasis. He also stated that intra-uterine death of the foetus was fairly common, and that clinical and pathological evidence pointed to malaria as an important predisposing factor in the toxaemias of pregnancy, such as pre-eclampsia, eclampsia and " nephritic " toxaemia.

While there is general agreement as to the harmful effect of malaria in the causation of abortions, miscarriages and stillbirths under epidemic conditions, there is considerable divergence of opinion in regard to the influence of the disease on pregnancy and on the newborn infant among the indigenous populations of highly endemic areas. In Sierra Leone, Blacklock and Gordon (1925*b*) found no evidence that malaria predisposed to abortion, but they had no doubt as to its harmful effect on the newborn infant, for out of 51 children born with infected placentas, 23 per cent. died within 7 days ; while of 93 born with the placenta non-infected, only 5 per cent. died within this period. Perves (1946) in the Cameroons and Weselko (1922) in Albania also considered malaria to be an important cause of infant mortality. Butler (1930) in the Gold Coast estimated that only 70 per cent. of infected placentas go to full term, while Welch (1924) in Kenya stated that he had observed many abortions due to malaria, which he considered might be caused by thrombosis in the vessels of the umbilical cord. Garnham (1938) suggested that many of the abortions in malaria were due to death of the foetus through physical

interference with the circulation of blood in the placenta (*i.e.*, because the intervillous spaces are filled with an almost solid mass of reticulo-epithelial cells), rather than to the direct effect of malarial toxins. He concluded that the incidence of abortion varies inversely with the degree of immunity in the mother, a view also expressed by Mary Blacklock (1941) as the result of her experience in West Africa. Garnham found little evidence of increased mortality in the first 7 days of life, and concluded that malaria was seldom a cause of stillbirth in Kisumu (Kenya), since out of 111 cases among mothers in that area, the stillbirth rate was only 1·8 per cent. Out of 124 women who aborted, only 19 (13 per cent.) were found to have parasites in the peripheral blood, whereas in a small group of non-immunes the percentage was nearly double. Schwetz and Peel (1934) in the Belgian Congo stated that in none of their cases did placental infection appear to exert a deleterious action on the child, and Bruce-Chwatt (1950) in Nigeria found no obvious relationship between malarial infection of the mother and abortion or premature birth.

It seems probable that the influence of malaria on the incidence of abortions, miscarriages and stillbirths and on the newborn child is very largely dependent on the degree of tolerance possessed by the mother.

Summary

1. Intra-uterine transmission of malaria from mother to child is a well established fact.

2. The manner in which the infection is transmitted remains a matter of conjecture, in spite of the many theories which have been advanced in explanation during the past 50 years.

3. Normally, the placenta provides a very effective barrier to the passage of the malaria parasite. Massive infection of the placenta can occur both in non-immune subjects and in those possessing a high degree of tolerance to malaria without the child becoming infected.

4. Congenital malaria can occur in the absence of demonstrable damage to the placenta. Infection can also be passed from mother to foetus before the onset of labour.

5. Congenital malaria can occur in the absence of any evidence of active malaria in the mother during pregnancy.

6. There is no evidence that infection may occur by passage through the placental barrier of sporozoites or of other exo-erythrocytic forms of the parasite.

7. The suggestion that the placental barrier may be broken down as the result of pathological changes due to malaria is probably correct, but is unsupported by concrete evidence.

8. The great majority of records relating to indigenous populations of malarious regions possessing a high degree of tolerance to malarial infection indicate that in such communities the incidence of congenital malaria is exceedingly low.

There is little statistical evidence as to the frequency of congenital malaria among non-immune subjects ; this has been estimated at from 1 to 4 per cent. where the mother has suffered from active malaria during pregnancy. In a series of 54 consecutive deliveries recently reported from Lagos, Nigeria, 4 cases were diagnosed as congenital malaria.

9. Congenital malaria can occur in association with any of the 3 common species of human malaria parasite, the relative incidence being probably dependent on the local frequency of each *Plasmodium*.

10. There is strong evidence that a considerable degree of temporary passive immunity to malaria can be, and frequently is, transmitted from mother to child.

11. Malaria in epidemic form is an important cause of abortion, miscarriage and stillbirth, and of deaths among newborn infants. There is some divergence of opinion as to its effects in this respect among indigenous populations of malarious regions. It seems probable that these vary inversely with the degree of tolerance to the disease possessed by the mother.

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MALARIA

PARROT, L. & CATANEI, A. Eléments de paludométrie. [**Means of measuring Malaria Incidence.**] *Anais Inst. Med. Trop.* Lisbon. 1949, Dec., v. 6, 327–49, 6 diagrams. English summary.

The authors describe in detail the various indices, based on the palpation and measurement of the spleen and on the microscopic examination of the blood, which have proved of value as measures of malaria endemicity, and in estimating the efficacy of control measures. The spleen index, the size of the average spleen, the splenometric index, the parasite index, the average intensity of parasitaemia, the plasmodimetric index (total and specific) and gametimetric indices, are all explained as are the methods of their evaluation as used by the authors in their work at the Pasteur Institute of Algeria. Examples are given of their value. *Norman White*

HUFF, C. G. [**Letter questioning Malarial Nature of the Exo-erythrocytic Bodies described by Professor Shortt and his Collaborators.**] *Trop. Med. News.* Bethesda, Md. 1950, June, v. 7, No. 3, 22–3.

In a letter to the Editor of *Tropical Medicine News*, Dr. Huff expresses doubt as to the malarial nature of the bodies described by Professor Shortt and his colleagues as pre-erythrocytic stages of *Plasmodium cynomolgi*, *P. vivax* and *P. falciparum*. These doubts are based on the following considerations :—

(1) A complete series of stages connecting the sporozoites with the erythrocytic forms in mammalian malaria has not yet been demonstrated.

(2) Controls were not run involving the biopsy of monkeys or human subjects inoculated with uninfected mosquitoes.

(3) In preparations treated with Zenker-formalin fixative and Maximow haematoxylin stain, Dr. Huff has found that the cytological appearance of the bodies described in mammalian malaria differs in certain respects from that presented by exo-erythrocytic forms of avian malaria, although the colour of the cytoplasm is the same in both cases.

Dr. Huff is careful to stress that his attitude is one of suspended judgment only, and that the opinions put forward in his letter are strictly personal.

As regards Dr. Huff's first point, although in mammalian malaria a complete series of stages connecting the sporozoites with erythrocytic forms has not yet been demonstrated, it is known to the reviewer that advances in this connexion have recently been made which extend previous knowledge, in that exo-erythrocytic bodies have been observed in the liver of a monkey as early as the third day after infection with *P. cynomolgi*. As regards the second point, which implies that the bodies described in mammalian malaria may have been produced by inoculation of mosquito tissue, massive quantities of uninfected mosquito tissue have more recently been injected into monkeys, accompanied by the biting of hundreds of the mosquitoes supplying this tissue, but no such bodies have been found in the liver, despite the most exhaustive search. Moreover, in any case, this objection can hardly be applied to the *falciparum* experiment, in which no mosquito tissue was inoculated, infection being by mosquito bite only. As regards the third point, material treated with Carnoy fixative and Romanowsky stain shows no such differences in cytological appearance between mammalian and avian exo-erythrocytic forms as have been described by Dr. Huff from sections treated by his method. In the reviewer's opinion, however, the most convincing proof of the validity of Professor Shortt's findings lies in the demonstration in the *falciparum* experiment of pre-erythrocytic forms in 3 stages of development after the subject had been exposed to the bites of

infected mosquitoes over a period of 3 successive days, exactly as foreshadowed by the results of Sir Neil Fairley's experiments on human volunteers at Cairns.]

G. Covell

Singh, J. & David, A. **Staining and Re-Staining of Oöcysts and Sporozoites from Infected Mosquitoes.** *Indian J. Malariology.* 1949, Dec., v. 3, No. 4, 349–52.

Oöcysts of *Plasmodium* in the mid-gut of mosquitoes may be stained with aqueous toluidine blue (0·1 per cent.), undiluted Leishman's stain (for 2 to 3 minutes), Giemsa's stain (20 per cent.), or J.S.B. stain (solution 1 for 2 or 3 minutes). The gut is fixed in the usual way with 10 per cent. formol saline, and after being stained, is dehydrated with xylol-acetone mixtures, cleared and mounted. Sporozoites can be readily demonstrated by use of the J.S.B. technique ; the infected glands are dissected in a very tiny drop of normal saline, crushed gently under a cover slip, the latter is lifted gently from the slide and both are preserved for staining after fixation in methyl alcohol. They are immersed in solution 1 for 4 or 5 minutes, washed in buffered water, dipped momentarily in solution 2, washed again and returned to solution 1 for a further 4 or 5 minutes. Detailed stages of the techniques are described.

P. C. C. Garnham

Fessler, K., Fischer, L. & Sorg, G. Anophelesfunde in Südwürttemberg und ihre Beziehung zum Problem der einheimischen Malaria. [**Anopheline Records in South Württemberg and their relation to Autochthonous Malaria.**] *Ztschr. f. Tropenmed. u. Parasit.* Stuttgart. 1950, Apr., v. 1, No. 4, 458–88, 9 figs. & 4 charts. [Numerous refs.]

The English summary appended to the paper is as follows :—

" 1. During the summer seasons of 1946–1949 systematical surveys for Anopheles were made in the rural region near Tuebingen, South Wuerttemberg. Nearly all villages were found inhabited to a small degree, anophelines were numerous only in the region of " Blaulach," an old bed of the river Neckar. Surveys extending over several years showed a gradual decrease of anophelines. In the whole region, river valley as well as surrounding heights, *A. typicus* is predominant ; only in the valley about 30% of *A. mac. messeae* are interspersed. *A. bifurcatus* was found in some breeding places near the woods of the Blaulach region.

" 2. The numerical relation of the two *maculipennis* races depends on the character of breeding places. *Typicus* is adapted to cultured lands, to small and shallow breeding places, mostly artificial, with scanty vegetation and subject to major changes of temperature. Additional *messeae* populations are found only in humid areas with large and densely grown-over breeding places, whose depth prevents extreme changes of temperature.

" 3. *A. bifurcatus* var. *missirolii* is mostly confined to stables.

" 4. There is reason to believe that mosquitoes of said region are reduced numerically in the course of time and that the numerical relation of the two *maculipennis* populations is shifting. After cultivation of soil and river regulation the *messeae* race seems considerably reduced whereas *typicus* still finds a bearable biotop on dry land with small breeding places. Moist years favour the development of *messeae ; A. mac. typicus* adjusts itself better to dry seasons.

" 5. It can furthermore be assumed that the decrease of endemic malaria during the second half of the 19th century is due to the general diminution of mosquitoes and also to shift in *maculipennis* populations. This explains

why in this region of Southern Germany which was formerly a focus of autochthonous malaria no fresh outbreaks have occurred in our days."

Smith, H. F., Dy, F. J. & Del Rosario, F. G. **Pictorial Keys for the Identification of Philippine Anopheline Larvae and Adults.** Reprinted from *Acta Med. Philippina*. 1950, Jan.–Mar., v. 6, No. 3, 195–8, 2 folding pls. [20 refs.]

Rey, H. & Renifo, S. *Anopheles* (*N.*) *nuñez-tovari* infectado en la naturaleza con Plasmodium Sp. [**Natural Infection of** *A. nuñez-tovari* **with Plasmodia.**] *Rev. Acad. Colombiana de Ciencias Exactas, Fisicas y Naturales*. 1950, May, v. 7, No. 28, 534–8, 7 figs. on 1 pl. [14 refs.] English summary.

In a malaria survey of Puerto Santander, on the Venezuelan frontier of Colombia, 65 persons were examined : the spleen rate was 57 per cent., the parasite rate 17 per cent. *Plasmodium vivax* was the commonest parasite, *P. falciparum* and *malariae* also being found. Six species of *Anopheles* were collected : *A. nuñez-tovari* was common in houses, and was the only one found infected. *P. A. Buxton*

i. Radosavljević, A. L'histamine et les antihistaminiques dans la pathologie du paludisme et de la tuberculose. [**Histamine and Antihistamines in the Pathology of Malaria and Tuberculosis.**] *Srpski Arkhiv za Tselukupno Lekarstvo* (*Arch. Serbes de Méd.*). Belgrade. 1950, June, v. 48, No. 6. [In Yugoslavic 377–84. [11 refs.] French summary 384–5.]

ii. ——. Le paroxysme fébrile palustre—phénomène allergique. **The Malarial Febrile Paroxysm—an Allergic Phenomenon.**] *Ibid.* [In Yugoslavic 447–51, 2 charts. French summary 451.]

The author's thesis is that febrile attacks in malaria should be interpreted as essentially allergic manifestations and not as the effects of a direct toxin from the parasite.

i. He distinguishes a "primary antigen" (the parasite) and a " secondary antigen " quite distinct from it, which is responsible for the secondary febrile attacks and is represented by disintegration products of infected red cells. The body sensitized to these products reacts to the next schizogony allergically and this is manifested clinically by the febrile attack.

On the principle that histaminic substances should thus be produced as a result of antigen-antibody reaction, the author has used synthetic anti-histaminic preparations, having solely anti-allergic properties, during the course of malaria and claims that on several occasions the febrile attacks were cut short and even prevented.

He points out that during the antihistaminic treatment, the parasite continued to multiply and this is given as evidence to support the thesis that the secondary febrile attacks could not have been due to a parasitic toxin, since the febrile attacks ceased, although the preparation used had no antipyretic or plasmodicidal properties.

The author develops an analogous argument relative to " granulomatous tuberculosis " and " destructive phthisis," the primary antigen being the tubercle bacillus and the secondary an allergen.

The therapeutic implications of these points are being pursued in the author's clinic.

ii. The author describes a case in which actual secondary febrile attacks were cut short or prevented in a patient with malaria, by the exclusive use of the synthetic antihistamines Antistine—Ciba and Théphorine—Roche. He

points out that these substances have no action other than an antihistaminic one, and he concludes that the case supports his hypotheses outlined above.

H. J. O'D. Burke-Gaffney

Casula, D. La funzionalità epatica nella malaria. [**Liver Function in Malaria.**] *Arch. Ital. Sci. Med. Trop. e Parassit.* 1950, Mar., v. 31, No. 3, 222–48. [23 refs.] English summary.

The English summary appended to the paper is as follows :—

" 55 partly chronic, partly acute malaria cases were investigated with regard to the glycaemic curve after introduction of glucose, the aminoacid curve of the blood after the introduction of glycocoll, the cholesterin curve after introduction of cholesterin, the Takata test and the bilirubin content of the blood with the purpose of determining the functional conditions of the liver.

" The three functional curves appeared to be more or less altered in many of the cases, the glycaemic curve showing a particular tendency to alteration. The Takata test was positive in a great number of the cases in accordance with other functional tests : this is, however, considered as a functional test of the reticulo-histocytary system which appears to be highly affected by malaria.

" The increased content of bilirubin in the blood which is often noted, particularly before and after the actual attack, depends upon the blood destruction and hyper-haemolysis from hyper-activity of the reticulo-histocytary system.

" The degree of deterioration of the liver depends to a certain extent on the seriousness and the stage of the infection : remarkable damage is noted in acute infections, particularly severe in chronic forms accompanied by frequent and recent relapses ; more rare and less severe in chronic forms in which the last attack occurred some considerable time back. Thus, the lesions of the liver tend to disappear gradually until complete recovery."

Coatney, G. R., Cooper, W. C., Ruhe, D. S., Young, M. D. & Burgess, R. W. **Studies in Human Malaria. XVIII. The Life Pattern of Sporozoite-Induced St. Elizabeth Strain Vivax Malaria.** *Amer. J. Hyg.* 1950, Mar., v. 51, No. 2, 200–215, 3 figs. [34 refs.]

This report is a summary of the chief characteristics of infections with the St. Elizabeth strain of *P. vivax* in 198 persons experimentally infected by mosquito bites during a period of four and a half years. The number of sporozoites in the salivary glands of each infected mosquito was graded from 1+ to 4+ ; the total number of pluses for the mosquitoes that bit each patient served as a rough measure of the infective inoculum. Drugs were used either as clinical prophylactics or therapeutically. Details concerning their administration have been given in previous papers of this series which have been summarized in this *Bulletin*. The drugs used were sulphadiazine, sulphapyrazine, quinine, mepacrine, pamaquin, SN1796, SN5241, SN6771, sontochin, SN7266, chloroquine, SN8557, metachloridine, colchicine, and pentaquine. Of these only pamaquin and pentaquine are classed as curative inasmuch as in combination with quinine they can prevent relapse.

Thick blood smears were examined daily during 45 to 60 days after exposure to infection and at least once weekly through the first 18 months.

Of the 198 subjects, 195 developed overt malaria. Of the 3 who failed to do so 2 were lost from observation before delayed primary attacks were due. Four hundred and twenty-nine attacks of malaria were observed in these 195

persons ; 135 attacks occurred within 2 months after mosquito bites, and 294 in a period of 6 to 14 months after bites.

Seventy-eight subjects received no prophylactic drug ; 75 of these developed patent malaria 11 to 20 days from initial exposure. The remaining 3 had primary attacks delayed to 298, 318 and 319 days after exposure ; all these three men had had relatively small inocula.

Of 120 men given protective medication, 41 had primary attacks in from 12 to 64 days after exposure : 76 had primary attacks delayed to from 193 to 350 days after exposure. Three never developed overt malaria [see above] The failure of drugs to prevent early primary attacks could be explained in all the 41 cases by the inadequacy of the regimen adopted.

The duration of latency after treatment of early attacks bore no relation to the drug used provided that the treatment was capable of eradicating erythrocytic parasites. Of the 116 men given antiplasmodial therapy during early primary attacks, 10 were treated with pentaquine and quinine for 14 days : none of the ten developed late attacks. Only one adequately observed patient treated with a non-curative drug failed to develop a late attack ; he was treated with quinacrine [mepacrine].

The mean time of onset of the attack after mosquito bites was in 74 delayed primary attacks 287·1 days ; in 79 relapses after early activity it was 275·6 days. Long-term latency was not dependent upon the month or season during which infection occurred, sporozoite dosage, degree or duration of early patent parasitaemia, or the nature of early non-curative therapy. During long-term latency subinoculation was negative and homologous strain superinfection could be accomplished. During the period of late activity several acute attacks usually occurred, the intervals between them being greater when slowly eliminated drugs were used. The total duration of late activity was much longer in untreated than in treated cases. It is unlikely that the final termination of overt malaria in the treated subject is a result of acquired immunity to erythrocytic parasites. *Norman White*

Cooper, W. C., Coatney, G. R., Culwell, W. B., Eyles, D. E. & Young, M. D. **Studies in Human Malaria. XXVI. Simultaneous Infection with the Chesson and the St. Elizabeth Strains of** *Plasmodium vivax*. *J. National Malaria Soc.* 1950, June, v. 9, No. 2, 187–90, 1 fig.

Fifteen white male volunteers in Texas were subjected to experiment. Nine were inoculated by mosquito bite with the Chesson and the St. Elizabeth strains of *P. vivax* simultaneously, three with the Chesson strain alone, three with the St. Elizabeth strain alone. Each man was bitten by ten infected mosquitoes. The Chesson strain usually produces an infection with several closely-spaced attacks, while the St. Elizabeth strain causes an early primary attack, several months of latency, and a series of late attacks in close succession. The volunteers remained under close observation for 18 months after infection. All malaria attacks were interrupted by a four-day course of treatment with chloroquine, begun on or before the 5th day of patent parasitaemia.

The men infected with either the Chesson or the St. Elizabeth strain alone developed the types of infection characteristic of these strains. Men with the double infections displayed recurrent activity throughout a period of nearly one year. There were exceptions ; one displayed a relapse pattern similar to that of the Chesson controls, and two had relapses similar to that of the St. Elizabeth controls. The average number of acute attacks per volunteer in the doubly-infected group was no greater than in the St. Elizabeth group and very little greater than in the Chesson group. *Norman White*

Siguier, F., Piette, Coblentz, B., Lemenager & Trélat. Réflexions sur certains paludismes pernicieux actuellement observés en France. [**Reflections on some Cases of Pernicious Malaria observed in France.**] *Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1949, Nos. 30/31, 1329–34.

The moral of this paper is that the possibility of malaria as a diagnosis should always be borne in mind when a patient who has recently returned from a malarious country presents anomalous symptoms. Four very remarkable cases are described in which failure to make a prompt diagnosis jeopardized the patient's life.

A parachutist after some months service in Indo-China returned to France by air. He was fêted by his comrades the day of his arrival and given a dinner at which he drank but two glasses of wine. On retiring for the night he had a headache and felt shivery. The following morning he was drowsy and in a confused mental condition : he had vomited. It was assumed that he was suffering from the effects of a " night out ". Later in the day he wandered about the hotel half-dressed and crazy. The police were called to remove this unwanted guest : fortunately they thought the patient might be ill. The first doctor called thought it to be a case of pneumonia in an alcoholic. A second medical opinion a day later suggested meningitis with a meningeal haemorrhage. Fortunately one of the authors was called in, 48 hours after the onset, when coma was almost complete : the patient's temperature was 40°C. Blood examination revealed numerous *P. falciparum* parasites. Recovery followed antimalarial treatment.

A young girl of 19 suffered from an attack of pleurisy in Equatorial Africa. She came back to France to convalesce. Here she had an attack of acute pain in the left chest and an oscillating temperature, with enlargement of the spleen. The X-ray of the chest revealed no abnormality but a diagnosis of disseminated tuberculosis was made. Two months' treatment with streptomycin followed. She showed no improvement. While under treatment another acute attack of fever developed with cerebral symptoms ; tuberculous meningitis caused by streptomycin-resistant bacilli was diagnosed though lumbar puncture had revealed no abnormality of the cerebrospinal fluid. It was the father of the patient who raised the question of malaria : he had been 30 years in the colonies. A heavy infection with *P. falciparum* was found. Recovery followed.

The third case was that of a patient who had been diagnosed as suffering from " septicaemia with psychic disturbance in an alcoholic ". He had recently been in Africa. He, too, had a *P. falciparum* infection which yielded to treatment.

A woman who had been a long time in the Ivory Coast fell ill in Nice. The symptoms led a neurosurgeon to suspect a cerebral tumour and the possibility of surgical intervention was seriously discussed. One of the authors was called in consultation. *P. falciparum* was found by sternal puncture but not in the peripheral blood. Complete recovery followed antimalarial treatment.

Norman White

Wahi, A. L. **A Case of Sciatica due to Malaria.** *Indian Med. Gaz.* 1950, June, v. 85, No. 6, 255.

Smith, H. F., Dy, F. J. & Cabrera, D. J. **Untoward Effects of Quinacrine Hydrochloride as observed in 302,206 Malaria Cases.** Reprinted from *Acta Med. Philippina.* 1950, Jan.–Mar., v. 6, No. 3, 185–90. [27 refs.]

The authors refer to the extensive literature dealing with the untoward effects of quinacrine [mepacrine] and which has been fully summarized in this *Bulletin* [see in particular, 1947, v. 44, 763].

The present paper deals with a study of 302,206 Filipino patients suffering from *P. vivax* (82·5 per cent.) or *P. falciparum* (17·5 per cent.) malaria. All were observed during treatment with mepacrine and for at least two weeks afterwards : most of them were ambulant, and those patients were excluded from the study who were not intelligent enough to cooperate either in describing symptoms or in following directions.

The drug used was quinacrine hydrochloride ("Atabrin," Winthrop) and the standard adult dose was 0·4 gm. t.d.s. on the first day and 0·1 gm. t.d.s. from the second to the seventh day. Treatment began on the first to the third day after onset.

The distribution of side-effects is shown in a table. In all 220 (0·073 per cent.) showed one or more untoward effects. Twenty-one conditions are listed, the commonest being nausea and vomiting (42 per cent.) ; anorexia (26) ; manic excitement or delusion (21) ; insomnia (15) ; epigastric discomfort (10·5 per cent.). Such conditions as vertigo, temporary loss of memory, talkativeness, headache and depression occurred in 5 to 7 per cent. of patients and the remaining effects were mostly 1 per cent. or less.

The untoward effects were temporary and disappeared in 1 to 8 days after discontinuation of the drug. In 73 per cent. of cases, these manifestations disappeared even when the drug was continued.

The authors discuss the difficulty in distinguishing between the effects of the drug and of the malaria itself and the careful attempts made to exclude the latter. Examples are given.

The authors conclude from these observations that quinacrine hydrochloride in the dosage described was attended with relatively few untoward reactions when it was given to these patients.

[While this paper adds little that is new to the recognized effects of mepacrine, it is a very valuable quantitative contribution in that it covers observations of nearly one-third of a million cases.] *H. J. O'D. Burke-Gaffney*

Brito, R. S. & Cotrim, J. Observações sobre o tratamento da malária pela cloroquina em Porto Velho—Guaporé. [**Observations on the Treatment of Malaria by Chloroquine in Pôrto Velho—Guaporé.**] *Rev. Brasileira Med.* Rio de Janeiro. 1950, Mar., v. 7, No. 3, 166–72. English summary.

This is a record of the treatment of 665 patients suffering from acute malaria with chloroquine in Guaporé, Brazil. The diagnosis was confirmed microscopically in all cases, the incidence of parasite species being *P. vivax* 320, *P. falciparum* 352 and *P. malariae* 81. The patients were of all age groups. They were admitted to hospitals in Porto Velho from the city and surrounding areas. Chloroquine diphosphate was given in tablet form, each containing 0·15 gm. of base. The total dose for adults of 60 kgm. weight and over was ten tablets, 4 for the initial dose, 2 eight hours later, and 2 on each of the second and third days. Tolerance was good and in no case were toxic symptoms sufficient to indicate cessation of treatment. Malaria symptoms disappeared 24 to 48 hours after treatment was begun. The blood of 304 patients was re-examined after the completion of treatment. No parasites were found in 81 per cent. In 22 of 135 *P. falciparum* infections gametocytes were found after treatment though schizonts alone had been found before. *Norman White*

Smith, H. F., Dy, F. J. & Cabrera, D. **Studies on the Efficiency of Chloroquine and Chlorguanide as Antimalarials : II. As Curative Agents against Philippine Strains of Plasmodia.** Reprinted from *Acta Med. Philippina.* 1949, Oct.–Dec., v. 6, No. 2, 137–52.

This paper relates to the second phase of an experiment carried out in two localities in the Philippines, San Felipe in Luzon Island and Kamingaoan in

Negros Island, with the object of assessing the relative efficacy of chloroquine and proguanil (chlorguanide) as antimalarials. The first phase, which was concerned with the relative suppressant value of the two drugs, was reviewed in this *Bulletin*, 1949, v. 46, 612.

For suppressive treatment the subjects of the experiment had been divided into 3 groups, Group I receiving chloroquine, Group II proguanil and Group III tablets containing sodium bicarbonate only. The subjects of the present study were 72 persons who developed overt malaria during the 12 months following the cessation of suppressive treatment, either for the first time or as relapses from previous attacks. Those in Groups I and II received as therapeutic treatment courses of the same drugs as had been used as suppressives in their respective group. Those in Group III were treated with quinine sulphate. The adult therapeutic dosage administered in each group was: Group I, chloroquine 2 tablets, each 0·2 gm. twice the first day and then 2 tablets once daily for 3 days (2·5 gm.). Group II, proguanil 2 tablets, each 0·10 gm. thrice daily for 3 days and then 1 tablet thrice daily for the 4 following days (3·0 gm.). Group III, quinine sulphate, 1 tablet, each 0·30 gm. 4 times daily for 5 days (6·0 gm.). Two months before the cessation of suppressive treatment, intensive mosquito control work was instituted in and around the camps where the experiment was conducted, in order to preclude as far as possible any chance of reinfection. Of the 72 subjects studied, 69 (95·8 per cent.) had *P. vivax* infection either alone or concurrently with either *falciparum* or *malariae*. In the case of mixed infections, *P. vivax* was invariably observed to survive the concomitant *Plasmodium*.

More relapses were observed among the proguanil group than among those treated with either chloroquine or quinine. No significant difference could be shown between the two groups in respect of the duration of the latent non-parasitic period or the interval between relapses. In any case, the numbers treated in each group were too small to admit of any very definite deductions as to the relative efficacy of the drugs used. A more interesting feature of the experiment was the different results achieved in San Felipe as compared with Kamingaoan. In San Felipe overt attacks occurred in both the proguanil and chloroquine groups in spite of suppressive and therapeutic treatment, whereas in Kamingaoan none of the originally positive subjects in either of these two groups showed parasites on subsequent examination. The relapse rate in those treated therapeutically with quinine was also higher in San Felipe than in Kamingaoan.

These observations led the authors to suppose that there may be inherent differences between the Kamingaoan and San Felipe strains of *P. vivax*. It is thought possible that the latter may have been introduced during the second world war by Japanese or American troops infected with resistant South-west Pacific strains of parasite in previous campaigns. *G. Covell*

CHAUDHURI, R. N. & RAI CHAUDHURI, M. N. ***Falciparum* Infection Refractory to Paludrine.** *Indian J. Malariology*. 1949, Dec., v. 3, No. 4, 365–9, 3 charts. [11 refs.]

A boy aged 12 years, with a history of several days' fever and with parasites of *P. falciparum* in the peripheral blood, was treated with proguanil 0·6 gm. by mouth, followed by 0·3 gm. daily for the next 8 days (total 3·0 gm.). There was no apparent effect on the fever or on the parasites, rings being found in the blood smears each day and crescents from the 10th day onwards. Both blood and urine gave a strongly positive reaction for proguanil. After an interval of 4 days a further course of proguanil was given, 0·6 gm. on the first day followed by 0·3 gm. for the next 3 days, but still with no apparent effect.

At this stage a volunteer was inoculated with the patient's blood and at the same time a single dose of chloroquine (0·5 gm.) was administered to the patient. The temperature promptly dropped to normal and parasites disappeared from the peripheral blood.

The volunteer was given proguanil 0·3 gm. daily for 4 days, but 17 days after inoculation he developed fever and parasites of *P. falciparum* were found in the peripheral blood. He was again given proguanil 0·3 gm. twice daily, but with no apparent effect on the pyrexia even after 8 days. As in the first case, the blood and urine gave a strongly positive reaction to the drug. At this stage his blood was inoculated into a second volunteer, who developed fever and parasitaemia 15 days later. He was treated with proguanil 0·2 gm. twice daily for 10 days, parasites disappearing from the peripheral blood within 48 hours and the temperature falling to normal on the 4th day. It is suggested that the positive response of this second volunteer may have been due to the fact that he had received 6 injections of acetylarsan as treatment for tropical eosinophilia. The first volunteer was cured with chloroquine.

The authors stress the fact that although strains of *P. falciparum* naturally resistant to proguanil exist in India, they are encountered only very exceptionally. *G. Covell*

Gaud, J., Schneider, J. & Mechali, D. Action comparée de la nivaquine et du chloriguane en prophylaxie collective du paludisme. [**Comparative Efficacy of Nivaquine and Chloriguane in Mass Prophylaxis of Malaria.**] *Bull. Inst. Hyg. Maroc.* 1949, v. 9, Nos. 1/2, 121–9.

These experiments were carried out in the Charb, an alluvial plain near the Atlantic seaboard of Northern Morocco. Two groups each consisting of three "villages" were chosen. The villages were as comparable as possible with respect to malaria endemicity and anophelism: each contained about 200 inhabitants. One village in each group was an untreated control. The inhabitants of the other two villages received prophylactic medication, once a week in one group, twice monthly in the other. The drugs administered were nivaquine [presumably C] and rhodopraequine in one group, chloriguane [proguanil] and rhodopraequine in the other. The tablets used each contained 0·1 gm. of nivaquine or chloriguane and 0·01 gm. rhodopraequine. The adult dose was 3 tablets; for those aged 6 to 12, two tablets; for lower ages, one tablet. The distribution of drugs was carried out from June to October inclusive in both 1947 and 1948. The results of the treatment were judged by variation in spleen, splenometric and parasite indices.

During the first year the protection afforded by the two drugs nivaquine and chloriguane on the weekly schedule was almost equal but during the second year nivaquine gave much better results. It seems possible that parasites developed resistance to chloriguane. The twice-monthly rhythm of distribution gave insufficient protection but such as it was chloriguane was more effective than nivaquine. This twice-monthly distribution was only carried out in one of the two years. *Norman White*

i. Singh, J. **Trials with Neochin in the Treatment of Simian Malaria.** *Indian J. Malariology.* 1949, Dec., v. 3, No. 4, 353–5.

ii. Chaudhuri, R. N. & Rai Chaudhuri, M. N. **A Note on Clinical Trials with Neochin.** *Ibid.*, 357–63, 3 charts.

i. Fifteen *rhesus* monkeys, infected with *Plasmodium knowlesi*, were treated with Neochin, a synthetic product derived from cinchonine with the absence of the methoxy (CH_3O) group of the quinoline base, claimed by the

manufacturers to be an antimalarial drug. The total dosage varied from 1½ grains (corresponding to that recommended for a human being on a body weight basis) to 30 grains (the actual dosage laid down for an adult human subject). The results up to a total dosage of 24 grains were poor, but with 30 grains parasites disappeared from the peripheral blood within an average of 82 hours. Even in this dosage, however, 4 of the 6 animals suffered relapses. No toxic effects were noted. It was concluded that in the treatment of *P. knowlesi* infections in monkeys the drug is inferior to quinine and to any of the synthetic antimalarials in common use.

ii. Clinical trials with Neochin were carried out on 7 cases of *P. falciparum* infection, 6 of *P. vivax*, 1 of *P. malariae* and 2 of mixed *P. falciparum* and *P. vivax*. Two régimes were used : (A) One tablet, 5 grains, twice daily for 2 days and once daily for 2 days, and (B) one tablet thrice daily for 4 to 5 days. Régime B was introduced because Régime A, which was that recommended by the manufacturers, proved inadequate.

Only 3 patients were treated with Régime A. One of these, a case of *falciparum* infection, not only failed to react, but became so seriously ill that after the 4th day chloroquine was administered, which resulted in prompt recovery. Thirteen patients were treated with Régime B, the average period of fever being 3·5 days, and the average period of parasitaemia 4·1 days. Eleven of these were observed for periods of 14 to 48 days. Seven of them (5 *falciparum*, 2 *vivax*) relapsed from 4 to 31 days after the completion of treatment. No toxic effects were noted.

It was concluded that Neochin, in the dosage recommended, or even in double that dosage, was unsatisfactory for the treatment of human malaria.

G. Covell

Coatney, G. R., Cooper, W. C., Culwell, W. B., White, W. C. & Imboden, C. A., Jr. **Studies in Human Malaria. XXV. Trial of Febrifugine, an Alkaloid obtained from *Dichroa febrifuga* Lour., against the Chesson Strain of *Plasmodium vivax*.** *J. National Malaria Soc.* 1950, June, v. 9, No. 2, 183–6, 1 fig. [12 refs.]

A medicinal herb Ch'ang Shan, has been in use in China for many centuries as a remedy for malaria [see this *Bulletin*, 1949, v. 46, 611]. It is prepared from the dried root of *Dichroa febrifuga*, an evergreen shrub found in India and in South-west China. Two interconvertible alkaloids have been isolated from the root, febrifugine and isofebrifugine : the former displays most activity against avian malaria. Schmidt (personal communication to the authors) also found febrifugine to be 100 times more active than quinine against blood-induced *P. cynomolgi* malaria in the monkey. The drug is highly toxic.

Two volunteers in Texas suffering from delayed primary attacks of malaria which followed the bites of ten *Anopheles quadrimaculatus*, heavily infected with Chesson strain *P. vivax*, were given febrifugine dihydrochloride, 1·25 mgm. of base every six hours. After four doses had been given the treatment had to be discontinued because of violent vomiting. Four other attacks of malaria in these and two other subjects were treated with half the above doses. Again nausea and vomiting were prominent symptoms but the patients were able to complete a 14-day course of treatment. The blood became free from parasites in 2 to 5 days and fever ceased, but parasites reappeared on the last day of therapy in 3 of the 4 patients and in the other on the 11th day after the completion of treatment. The emetic action of the drug makes its use impracticable as a remedy for malaria.

Norman White

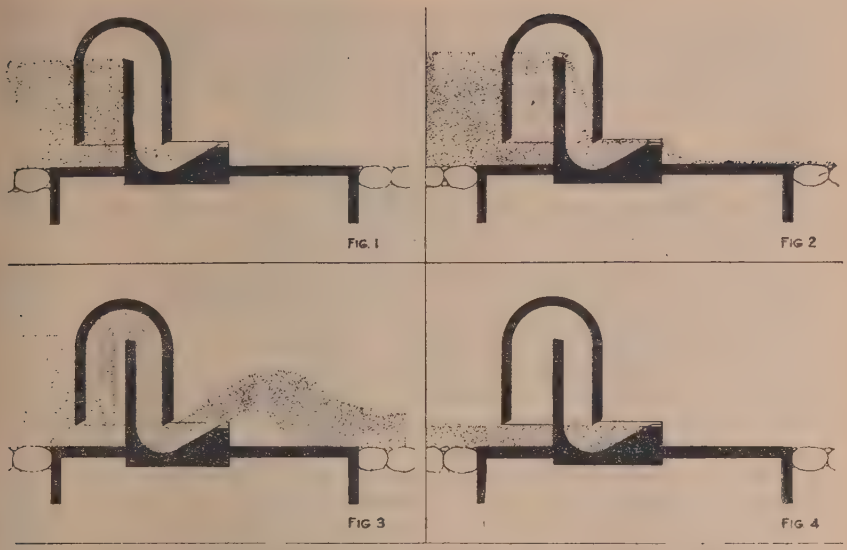
BANERJEA, R. **The Control of Malaria in a Rural Area of West Bengal.** *Indian J. Malariology.* 1949, Dec., v. 3, No. 4, 371–86, 1 chart.

This paper records the result of an experiment undertaken in a village in Western Bengal, with a population of 406 persons, to assess the value of proguanil in the prophylaxis of malaria. A second village with a population of 308 persons served as a control. At the commencement of the experiment the parasite rate in the experimental village was 5·6 per cent. and the spleen rate 47·6 per cent. The corresponding figures for the comparison village were 2·7 per cent. and 50·2 per cent. respectively. The experiment extended over two malaria seasons. During the first of these, proguanil was administered once weekly in a dosage of 1 tablet (100 mgm.) to adults, half a tablet to children from 2 to 12 years of age, and one quarter of a tablet to infants below 2 years. The figures relating to malaria cases, spleen and parasite rates showed little difference between the two villages during this period and it was concluded that the administration of this dosage once weekly was insufficient. During the second season the drug was administered twice weekly at intervals of 3 or 4 days. The spleen rate in the experimental village fell from 37·4 per cent. to 15·4 per cent. and the parasite rate from 5·3 per cent. to 0·3 per cent. during this period. In the comparison village the spleen rate increased from 33·4 per cent. to 46·8 per cent., while the parasite rate remained unaltered at 10·2 per cent. It was concluded that in order to secure satisfactory results, bi-weekly administration of the drug was essential.

The cost of drugs alone for such a programme was estimated at Re. 1-5-9 *per caput* per annum. This by itself represents a greater sum than has been allowed for by the Indian Health Survey and Development Committee for *all* health activities. When the cost of personnel and other items of administration is added, it becomes evident that the prophylaxis of malaria by this means in rural India is altogether outside the realm of practicability. *G. Covell*

DY, F. J., BERNARDO, B. C. & BERNARDINO, C. E. **The Smith Automatic Siphon.** Reprinted from *Acta Med. Philippina.* 1950, Jan.–Mar., v. 6, No. 3, 219–41, 6 pls. & 2 figs.

In the Philippines the well-known automatic siphon as a means of flushing streams is used as a measure of malaria control. Difficulties in construction and operation include percolation of water under the base of the dam, silting above the dam, bank erosion and by-passing of the siphon and clogging of the siphon with debris ; deliberate blocking or interference with the incorporated auxiliary siphon or priming vent prevents the operation of the main siphon. The authors designed the Smith Automatic Siphon with these difficulties in mind. There are no auxiliary and vent pipes. The cross-section area of the lower limb is smaller than that of the upper limb and its width from the central wall is between 15 and 18 inches ; this ensures that overflow of water from the upper limb strikes the water seal as close to the outer lip as possible, and in the ensuing siphonic action considerable velocity and turbulence are achieved. The clearance between the outer lip and the surface of the water seal is half to two-thirds of an inch, created by a slit 2 inches wide in the front edge of the seal ; this clearance is obliterated by the wave action caused by falling water, with which air bubbles escape through the seal and so promote the siphonic flush. The diagram illustrates the action of the siphon and its simplicity of construction. Complete plans and specifications are included in the paper. Reinforced cut-off walls and apron are provided above and below the dam; vertical steel bars protect the inlet of the siphon ; a strong revetment wall



Steps in the operation of the Smith Automatic Siphon

Fig. 1. Reservoir full, water about to flow over the crest. Clearance below lip of lowér limb is patent.
Fig. 2. Water flowing over the crest, sealing lip of lower limb. Air in chamber being carried out with the overflow.
Fig. 3. Siphon action in full operation.
Fig. 4. End of flushing. Air has entered the chamber.

[Reproduced from *Acta Medica Philippina*]

is constructed ; wide silt gates are placed in the dam wall. The efficiency of the siphon is enhanced by modification of the crest in the chamber to slope to the centre and towards the lower limb ; by this means a jet of water falls at the lower lip even in the dry season, rather than an even trickle over the full width of a level crest. The process and continuity of construction of the dam and the incorporated siphon are briefly described. *R. Ford Tredre*

Cavaillon, A., Adam, J. P., Bernard, L., Camain, R., Jaujou, C. & Trinquier, E. Deux années de lutte contre le paludisme en Corse : premiers résultats. [**First Results of a Two Years Antimalarial Campaign in Corsica.**] *Bull. Acad. Nat. Méd.* 1950, v. 134, Nos. 15/16, 320–25.

A malaria survey of Corsica was carried out in 1947 by Toumanoff & Rageau [this *Bulletin*, 1950, v. 47, 104]. In March 1948 the reorganized Antimalaria Service began the systematic treatment of dwellings and outhouses with DDT spray. During that year the measure was restricted to the south-western part of the east coast and a small part of the west coast : 17,081 rooms accommodating 11,929 persons were treated. The spectacular disappearance of insects from houses made the measure popular. A remarkable decline in malaria incidence followed. In March 1949 the spraying was extended to cover most of the malarious regions in the island. The results as regards anopheles were again very favourable but the appearance of DDT-resistant flies excited adverse comment. In future it is proposed to combine Octa-chlor with the DDT. In 1949 the protected population numbered 47,472. In 1949 only 76 positive

blood smears were found, a fraction of previous years figures. There was only the usual spring wave of malaria ; the usual later *P. falciparum* wave failed to materialize. The total number of cases of malaria was but one-twentieth of the 1947 total. The prospects of liberating Corsica from malaria are bright.

Norman White

Mosna, E. & Alessandrini, M. La lotta antianofelica con DDT nella provincia di Latina nel 1948 e 1949. [**Campaign against *Anopheles* with DDT in the Latina Province during 1948 and 1949.**] *Riv. di Parassit.* Rome. 1950, Mar., v. 11, No. 1, 13–26, 9 graphs. English summary.

Experience in the Province of Latina in the years 1945–47 had led the authors to believe that in Mediterranean countries, where mosquitoes of the group *A. maculipennis* are the chief vectors, the rational use of DDT against the adult insect is, by itself, able to suppress malaria transmission. The continuation of the programme which is described in this report, for the years 1948 and 1949, has confirmed this belief. In 1948 spraying was carried out between March and the end of June : in 1949 it was started a month later. The quantities of DDT used were 1·90 gm. per square metre in 1948, 1·83 gm. in 1949, sufficient quantities to obtain residual action for about a year. Buildings treated included 153,043 rooms, 11,999 huts and 31,804 animal houses. In fixed catching stations not a single *A. maculipennis labranchiae* has been found since March 1949 in spite of diligent search. The number of clinical attacks of malaria reported declined rapidly from the beginning of the DDT campaign to attain negligible numbers in 1948 and none at all in 1949. The results of blood examinations show that the last positive *P. vivax* film was seen in September 1948. *P. falciparum* has not been seen since September 1947. No death has been attributed to malaria during the last 3 years. Formerly atebrin [mepacrine] tablets were distributed on a large scale ; not one was distributed in 1949. The cost of the DDT campaign was 322·70 lire per head of population in 1948, and 187 lire in 1949. The authors think that considerable economies could be made if DDT emulsions were used in place of the much more expensive solutions.

The DDT spraying was combined with the use of Octa-Klor to eliminate DDT-resistant house flies. The results of this endeavour are reported separately.

Norman White

Cambournac, F. J. C. Sobre o combate ao sezonismo no território de Goa (Estado da India). [**Antimalaria Campaign in Goa, India.**] *Anais Inst. Med. Trop.* Lisbon. 1949, Dec., v. 6, 7–39, 1 coloured pl. & 42 figs. on 12 pls. English summary.

Old Goa is situated some 10 kilometres from New Goa, the capital of this Portuguese territory in India. Only ecclesiastical monuments and buildings remain to remind one of the past worldly importance of old Goa, which once harboured a population of some 300,000. Today the permanent residents are few other than the inmates of religious houses, but thousands of pilgrims visit the old city and spend a night or more there. Epidemic disease appears to have been responsible for the desertion of Old Goa and the creation of a new capital in a healthier place. As to the nature of that disease there are several legends ; probably it was malaria. Malaria is hyperendemic in Old Goa ; in 1935 the spleen index of its inhabitants was 80 per cent., whereas in quite nearby places malaria is but little in evidence, with spleen rates of only 3 per cent. The enormous number of wells in Old Goa afford facilities for the prolific breeding of mosquito vectors ; there were formerly 3,500 wells.

The important malaria vectors in Old Goa are *A. fluviatilis*, *A. varuna* and *A. minimus* ; the last-named is, however, rare. *A. hyrcanus* var. *nigerrimus*

is of less importance *A. barbirostris, A. vagus, A. subpictus* and *A. jamesi* occur but are not vectors. The season of maximum transmission is from October to December, after the rains.

Control measures which were put in hand in 1948 included the distribution of paludrine (proguanil] to all residents, the closing of wells, clearing of shrubs and low vegetation, the cleansing and regularization of ditches, the demolition of ruined buildings and the spraying of houses with DDT. There has since been a decrease in the incidence of malaria. Larvivorous fish and DDT are to be used to control breeding in collections of water that cannot be eliminated. DDT spraying in churches, galleries and museums is not possible owing to the height of the walls and the value of mural decorations. Details of the work being done are given and the report is illustrated with numerous photographs including some of important buildings. *Norman White*

ADHIKARI, A. K. & GANGULI, N. **Malaria Control on Bengal-Nagpur Railway from 1925 to 1948.** *Indian J. Malariology.* 1949, Mar., v. 3, No. 1, 1–37, 1 map & 10 charts. [20 refs.]

This paper summarizes the activities of the Malaria Section of the Medical Department of the Bengal-Nagpur Railway from its inception in 1925 to the close of 1948. Throughout the greater part of that period a succession of important contributions to malaria literature by Senior WHITE and his colleagues made that work familiar.

A map shows that the area served by this railway includes the delta of the Hoogly River where *Anopheles sundaicus* is the vector ; the Midnapore District of Western Bengal (*A. philippinensis*); the coastal plain of Orissa (*A. annularis* and to a much less degree *A. aconitus*) ; the Chilka Lake area of Orissa (*A. sundaicus*) ; North Madras Coast area extending to and including Vizagapatam (*A. stephensi* and *A. sundaicus*) ; Jeypore Hill tracts (*A. fluviatilis A. varuna* and *A. minimus*) ; Eastern Satpura Ranges (*A. fluviatilis, A. varuna* and *A. culicifacies*) ; Hazaribagh Ranges and Ranchi Plateau (*A. fluviatilis* and *A. culicifacies*) ; Singhbhum Hills of Chota Nagpur, Bihar (*A. fluviatilis, A. varuna* and *A. minimus* ; *A. culicifacies* of no importance).

The malaria morbidity rate of the protected population showed a remarkable decline from 1945 to 1948 as did the number of anophelines captured in fixed catching stations. Many control measures have been used but it would seem that residual DDT spraying and the distribution of paludrine [proguanil] as a prophylactic both contributed much to the success achieved. In 1948 the *per caput* cost of malaria control measures was Rs.3.1. *Norman White*

SINGH, J., RAY, A. P. & NAIR, C. P. **Preliminary Investigations on the Chemotherapeutic Activity of Atebrin, Paludrine, Resochin, Camoquin, Metachloridine and Aphacrine on Simian Malaria.** *Indian J. Malariology.* 1949, Dec., v. 3, No. 4, 387–403. [29 refs.]

A total of 164 *Silenus rhesus* monkeys weighing 3 to 5 kgm. infected with *P. knowlesi*, which almost invariably produces a fatal infection, have been used in comparing the antimalarial potency of the substances named in the title [the constitution of aphacrine is not indicated] during the course of a wider investigation on indigenous and synthetic antimalarials. [For similar quantitative studies see RICHARDSON *et al.* (this *Bulletin*, 1947, v. 44, 289).] Healthy animals were inoculated intraperitoneally with blood from a donor monkey infected with *P. knowlesi*. The progress of the infection was followed in stained thick and thin blood smears and oral treatment was started with different dosage regimens of each drug, and lasted from 2 to 7 days or longer. The

effects of the drug on host and parasite were closely observed. The blood of those animals which became parasite-free was examined daily for a period of at least 3 months. Eradication of infection was verified by giving adrenaline, by sub-inoculation, superinfection or splenectomy. The peripheral blood was freed from parasites by all the drugs, most rapidly by chloroquine and camoquin (48 hours on average), and most slowly by metachloridine (144 hours). Four of these drugs apparently gave rise to radical cure, the minimum doses required being 100 mgm. daily for 7 days with atebrin [mepacrine], 100 mgm. thrice daily for 7 days with paludrine [proguanil], 250 mgm. daily for 3 days with chloroquine and 100 mgm. daily for 3 days with camoquin. On the basis of dosage of the free base, camoquin appears to be the most effective. Paludrine did not show up so favourably as in the treatment of human and bird malarial infections. *J. D. Fulton*

Schneider, J. & Schneider, C. R. *Pl. berghei* : absence " d'incubation " au cours du passage de souris à souris par voie intrapéritonéale. [**Absence of Incubation Period in** *P. berghei* **Infections passaged through Mice by the Intraperitoneal Route.**] *Bull. Soc. Path. Exot.* 1950, v. 43, Nos. 5/6, 324–32.

From the reports of various authors it would appear that the incubation period of *Plasmodium berghei* after blood-inoculation of its rodent host may vary from one to four days. However this variation is not due to the existence of a pre-erythrocytic development, since the present authors failed to detect any EE stages during the incubation period. They regard the latent phase merely as the time required for the small number of parasites introduced intraperitoneally to multiply by schizogony in sufficient numbers for their detection in the blood of the recipient host. In order to verify this hypothesis in the case of mice, the authors resorted to isodiagnosis, which enables one to determine the infection of an animal at any given moment by injecting 0·01 cc. of its blood intraperitoneally into a clean mouse. The resulting infection in the recipient indicates the presence of parasites in the blood of the donor at the time when its blood was taken.

By using this method in a series of experiments, in which mice were infected by the standard technique described previously [this *Bulletin*, 1950, v. 47, 1066], the authors have demonstrated that in passages of *P. berghei* the blood of the recipient mouse becomes infective within one hour (sometimes even in 10 minutes) after having been inoculated intraperitoneally with infected blood, the time corresponding to the speed with which the parasites multiply in the peritoneal cavity and penetrate into the circulation. Under these conditions, there would appear to be no true " incubation period " in the sense of a latent phase. It was also shown that the infection ran the same course and was invariably lethal for the mouse, irrespective of the period when the infecting blood was taken from the donor and of the number of parasites inoculated initially. *C. A. Hoare*

Galliard, H. & Lapierre, J. Modifications hématologiques au cours de l'infection à *Plasmodium berghei* chez la souris. Essais de diverses substances non spécifiques. [**Blood Changes in Mice infected with** *P. berghei*.] *Bull. Soc. Path. Exot.* 1950, v. 43, Nos. 5/6, 317–23.

The authors have studied the blood changes taking place in mice in the course of infections with *Plasmodium berghei*, and have attempted to influence the anaemia by administration of various non-specific substances. Experiments carried out on 110 mice have shown that the changes in the blood increase

with the duration of the disease, in the course of which there is a progressive development of anaemia, the number of red blood corpuscles dropping to 1 million per cmm. As regards the leucocytes, their number may rise from 10,000 (normal) to 120,000 per cmm., with an increase of the proportion of neutrophil granulocytes up to 78 per cent., as compared with the normal 25 per cent.

With a view to increasing the host's resistance, by inhibiting the multiplication of the parasites or by reducing the anaemia, the mice were treated prophylactically and curatively with para-amino salicylic acid, vitamin B_{12} and folic acid, but none of these substances, except the last-named, produced any appreciable results. Folic acid, given in daily doses from 1 to 2 mgm. (diluted in 0·5 cc. water), prolonged the life of the infected mice for several days but failed to reduce the anaemia or the number of leucocytes.

C. A. Hoare

GALLIARD, H. & LAPIERRE, J. Effets de la splénectomie sur l'évolution et les rechutes de l'infection à *Plasmodium berghei* Vincke et Lips chez le rat blanc. **Effect of Splenectomy on the Course and Relapse of Infection with *P. berghei* in the Rat.** *C. R. Soc. Biol.* 1950, Mar., v. 144, Nos. 5 6 402–3.

It is known that in adult rats infection with *P. berghei* usually runs a benign course with spontaneous recovery in a month, in contrast to mice, in which the infection is acute and mortal. In a previous paper one of the present authors has shown that in splenectomized rats the disease becomes indistinguishable from that in mice. In the present investigation the object was to find out whether splenectomy performed on rats recovered from an infection would provoke in them a relapse, and, if so, whether the course of infection would be as acute as in rats splenectomized before the inoculation of the parasites. Three rats were operated upon 5, 10 and 20 days respectively after the apparent disappearance of the parasites from the circulation. In all these cases there was a relapse 4 to 5 days after the operation, but the number of parasites did not exceed that found in primary infections and the infection ran a benign course, ending in recovery 23 to 25 days after splenectomy. In one rat there were two more relapses at weekly intervals. It was thus shown that the resistance acquired during the primary infection prevented the development of an acute infection, of the type provoked in rats splenectomized before the inoculation.

The following observations were made on the leucocyte count: in rats infected with *P. berghei* there is an increase in the number of neutrophil granulocytes, which may reach 35 to 70 per cent. as compared with 20 per cent. in normal rats, while the total number of leucocytes may rise from 12,000 (normal) to 24,000. In non-infected splenectomized rats the percentage of neutrophil granulocytes may fall to 10 five days after the operation and to 7 after 25 days. In recovered rats subsequent splenectomy causes a rise in the proportion of the neutrophil granulocytes, but it remains lower than in unoperated infected rats. In rats recovered both from primary and relapse infection the leucocyte count returns to normal. The changes in the formula correspond to the degree of infestation of the red blood corpuscles in the peripheral circulation.

C. A. Hoare

THURSTON, June P. **The Action of Antimalarial Drugs in Mice infected with *Plasmodium berghei*.** *Brit. J. Pharmacol. & Chemotherapy.* 1950, Sept., v. 5, No. 3, 409–16, 1 fig. [12 refs.]

VINCKE & LIPS (this *Bulletin*, 1948, v. 45, 979) first described *Plasmodium berghei* a parasite of certain wild rodents in the Belgian Congo which readily

causes severe and fatal infections in laboratory rodents. The value of mice infected with this parasite for the screening of potential antimalarial drugs has been investigated on a limited scale [this *Bulletin*, 1950, v. 47, 324, 325]. The present author has used a few drugs for a similar purpose. Mice were injected intraperitoneally with infected blood containing 5 to 15 million parasites and drug treatment once daily by the oral route was begun a few hours later and continued for 4 days. On the day after the last dose of drug and again two days later, stained films were used to determine the percentage of red blood cells infected. Examination of the blood of mice which had shown a response to treatment was continued twice weekly till parasites were again seen or otherwise till the 60th day after inoculation. If parasites were absent till the last examination the host blood was inoculated into fresh mice to find if a latent infection was present. The results indicated that *P. berghei* was less sensitive than *P. gallinaceum* to quinine or pamaquin [plasmoquine], about equally sensitive to proguanil [paludrine], more sensitive to mepacrine [atebrin] and chloroquine and very much more so to sulphonamides, and thus more closely resembles certain mammalian parasites. The activity of sulphadiazine was inhibited by *p*-amino-benzoic acid. *J. D. Fulton*

McGhee, R. B. **The Ability of the Avian Malaria Parasite, *Plasmodium lophurae*, to infect Erythrocytes of Distantly Related Species of Animals.** *Amer. J. Hyg.* 1950, July, v. 52, No. 1, 42–7, 1 fig.

The author [this *Bulletin*, 1949, v. 46, 912] had already succeeded in infecting mouse erythrocytes with *Plasmodium lophurae* in the circulation of chicken embryos. Now he describes the results of experiments planned to observe (1) the course of the infection and (2) the different species of animal blood that might prove to be susceptible. Ten-day embryos were used and parasites were inoculated intravenously. The day after this injection, 0·2 cc. of a foreign erythrocyte suspension was introduced. The blood of 14 different mammals was tried, also that of a lizard and a frog. The corpuscles of man, mouse, rabbit and pig became infected but mouse corpuscles were the only ones parasitized in any number (up to 3 per cent.). The morphology of *P. lophurae* in the mammalian corpuscle is described. The trophozoites tended to assume band forms or to possess cytoplasmic filaments ; there was early concentration of pigment ; after 36 hours, segmenting forms with 6 to 11 merozoites were found which occupied about four-fifths of the cell. No gametocytes were seen. These observations relate chiefly to parasites inside mouse corpuscles ; rabbit and pig cells disintegrated so quickly that only early development of parasites could be traced while human cells showed such a high degree of resistance to infection that only a few parasites were seen in them. *P. C. C. Garnham*

Taliaferro, W. H. & Taliaferro, Lucy G. **Reproduction-inhibiting and Parasiticidal Effects on *Plasmodium gallinaceum* and *Plasmodium lophurae* during Initial Infection and Homologous Superinfection in Chickens.** *J. Infect. Dis.* 1950, May–June, v. 86, No. 3, 275–94, 10 figs. [26 refs.]

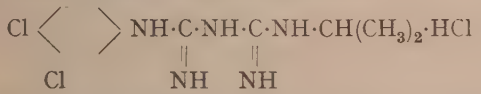
This is a continuation of the comparative immunological studies on *Plasmodium gallinaceum* and *P. lophurae* in chicks published previously [this *Bulletin*, 1947, v. 44, 40]. In the present work the authors have investigated the initial infection and homologous superinfection (in both cases blood-induced) with special reference to the number of merozoites produced and the viability of the parasites. The technique was the same as that described earlier [*loc. cit.*]

The experiments and observations are discussed in great detail, which does not lend itself to a short review. The main conclusions are as follows. In both species of parasites the asexual cycle in initial infections has a periodicity of 36 hours, but they differ considerably in the synchronism of the cycle, in the rate of reproduction, and in survival and death of the parasites. *P. gallinaceum* resembles more closely *P. cathemerium* and the parasites of Asian monkeys (*P. cynomolgi* and *P. knowlesi*), whereas *P. lophurae* is more like *P. brasilianum* and *P. floridense*. While in *P. gallinaceum* infections acquired immunity developed progressively, in *P. lophurae* it appeared suddenly at the peak of the infection. In the case of both these species immunity to homologous superinfection was chiefly parasiticidal, affecting all the erythrocytic stages. To some extent it also inhibited reproduction, as shown in a reduction of the merozoite mean per schizont and in a prolongation of the asexual cycle.

When latently infected chickens were superinfected with optimum numbers of normally developing parasites, the merozoite mean reached a lower value earlier, degenerate (crisis) forms appeared sooner, and the asexual cycle was more prolonged than in initial infections. It would appear that these reproduction-inhibiting effects are due to fundamentally parasiticidal mechanisms (antibodies). After a high degree of immunity has been acquired, the few surviving parasites become resistant to both parasiticidal and reproduction-inhibiting mechanisms, but non-resistant forms in each brood are rapidly removed from the blood. *C. A. Hoare*

Curd, F. H. S., Davey, D. G., Hendry, J. A. & Rose, F. L. **N^1-3 : 4-Dichlorophenyl-N^5-*Iso*propyl Diguanide—a Derivative of Proguanil Highly Active in Avian Malaria.** *Brit. J. Pharmacol. & Chemotherapy.* 1950, Sept., v. 5, No. 3, 438–44.

The substance named in the title has the formula—



—and thus differs from proguanil [paludrine] only in having one extra Cl atom in the benzene ring. It is a white crystalline powder soluble to the extent of 1 per cent. in water, and the solution is not decomposed by boiling. Tests were made against sporozoite- and blood-induced infections of *P. gallinaceum* in chicks and *P. relictum* in canaries, the drug usually being given orally. The results indicated that it was much more active on a weight basis than proguanil in *P. gallinaceum* infections, but it was about 7 times as toxic to ducks. The new substance was also much more active than proguanil against both types of infection with *P. relictum* and proved less toxic to canaries. Thus 0·05 to 0·1 mgm. per 50 gm. in chick infections and about double this amount in canaries controlled blood infections. Dosage of 3 to 4 times these amounts similarly controlled sporozite infections when treatment was begun within a few hours after inoculation. It was not found possible, however, to cure established infections of either parasite. For mice and rats the two substances were equally toxic, whereas proguanil was less toxic to dogs and monkeys. In dogs a dosage of 10 mgm. per kgm. by mouth twice daily for 5 days caused an acute haemorrhagic gastro-enteritis and death, whereas monkeys tolerated half this amount. The marked action of the drug against exoerythrocytic forms may prove of value in the tests now being carried out in human infections. *J. D. Fulton*

SINGH, J., RAY, A. P., NAIR, C. P. & BASU, P. C. **Screening of some Biguanide Derivatives for Antimalarial Activity.** *Indian J. Malariology.* 1949, Dec., v. 3, No. 4, 405–12.

A number of substances with structure based on that of paludrine [proguanil] have been tested for antimalarial properties in *P. gallinaceum* infections of chickens and in *P. knowlesi* infections of monkeys, by oral administration. The variations in the paludrine molecule consisted in the substitution of Br for Cl, or alteration of the position of Cl atom in the benzene ring, or by substitution at N^5 with the sulphanilamide, sulphathiazole or thiazole, sulphadiazine, sulphamerazine, and sulphamezathine grouping, while another substance contained the metachloridine grouping. The substance designated bromoguanide, in which Br replaces the Cl atom of paludrine, gave the only promising results in chicken malaria. In monkey malaria the latter compound and that isomer of paludrine with the Cl in the meta position gave the best results. Those with the sulphonamide compounds were not encouraging. *J. D. Fulton*

GREENBERG, J. & RICHESON, Edna M. **Potentiation of the Antimalarial Activity of Sulfadiazine by 2, 4-Diamino-5-Aryloxypyrimidines.** *J. Pharmacol. & Exper. Therap.* 1950, Aug., v. 99, No. 4, Pt. 1, 444–9. [16 refs.]

"1. Twenty-two compounds were tested for their ability to potentiate the antimalarial activity of sulfadiazine against *Plasmodium gallinaceum* in the chick.

"2. Only the two 2,4-diamino-5-aryloxypyrimidines were able to potentiate sulfadiazine activity in this infection. One quarter of the minimum effective dose of the diaminopyrimidines plus one-sixteenth of the minimum effective dose of sulfadiazine was the minimum effective regimen when these compounds were administered concurrently.

"3. The antimalarial activity of the diaminopyrimidines was partially inhibited by large oral doses of pteroylglutamic acid."

TRYPANOSOMIASIS

FAIRBAIRN, H. & CULWICK, A. T. **Distribution of Trypanosomes in Blood Films.** [Correspondence.] *Trans. Roy. Soc. Trop. Med. & Hyg.* 1950, July, v. 44, No. 1, 145–6.

The authors have previously [this *Bulletin*, 1947, v. 44, 649] described a method for determining the electric charge of trypanosomes by noting their position in relation to the erythrocytes in a stained film. This method was criticized by HORNBY [this *Bulletin*, 1949, v. 46, 811]. In the present paper the authors answer these criticisms.

In the first place, they state that the charge of the trypanosomes can already be demonstrated by their movements in relation to the red cells in a fresh blood film. Then they point out that the discrepancy in the results obtained by themselves and by Hornby may be due to the use of a different animal (guineapigs instead of rats) by the latter. And finally, they maintain that their interpretation of the charge of trypanosomes in stained blood films can be corroborated by measuring the trypanosomes : the length of those in contact with erythrocytes corresponds to that characteristic of positively charged forms, while those not touching the red cells have the measurements of

negatively charged forms. Anyone interested in further particulars of this controversy should read the original paper and notes. *C. A. Hoare*

See also p. 1222, COLONIAL OFFICE, **Studies in Mental Illness in the Gold Coast** [TOOTH, G.].

i. MONNET, A. & LAPEYSSONNIE, L. Association Mel B et Novocaïne. [**Mel B and Novocaine Mixtures (for Trypanosomiasis).**] *Méd. Trop.* Marseilles. 1950, Mar.–Apr., v. 10, No. 2, 306–11, 1 chart.

ii. —— & ——. Note sur le mélange Mel B/Novocaïne en solution dans la diméthylacétamide. [**Mel B and Novocaine Mixtures in Dimethylacetamide Solution.**] *Ibid.* 312–14.

iii. —— & ——. Association Mel B/acide para-aminobenzoique. [**Mel B and *p*-Aminobenzoic Acid Mixtures.**] *Ibid.* 315–18.

i. A patient critically ill with arsenical encephalopathy consequent on treatment by Mel B was restored to health by injections of novocaine (10 cc. of a 1 per cent. solution intravenously and 20 cc. subcutaneously). The novocaine had been given on the theory that (*a*) it might relieve the symptoms of derangement of the autonomic nervous system, and (*b*) it might serve as a source of *p*-aminobenzoic acid, a substance which is essential to the body's metabolism, disorganized by the arsenic or B.A.L. contained in the Mel B.

The authors thereupon decided to investigate preparations of Mel B in association with novocaine, in the hope that such preparations might be less toxic than Mel B alone. It was found possible to obtain 3·6 to 5·0 per cent. solutions of novocaine in 3·6 per cent. Mel B in propylene glycol. The two drugs were shown to be present as true mixtures in such solutions without any chemical combination, and appeared to be stable for several weeks if kept in well-stoppered bottles in the dark.

Preliminary tests for toxicity and trypanocidal activity were carried out in goats, sheep and dogs. The mixture was then injected intravenously without untoward incident in a man weighing 60 kgm. in the amounts of 1·7 cc. and 3·4 cc. (*i.e.*, 1 mgm. and 2 mgm. of Mel B per kgm. body weight).

The trypanocidal effects in animals were regarded as satisfactory, but the combination has not yet been tested against a human infection.

ii. It is possible to obtain 3 to 5 per cent. solutions of novocaine in 3 per cent. Mel B in dimethylacetamide. As with the preparations in propylene glycol (see (i) above) the two drugs were shown to be present as true mixtures without chemical combination, and to be reasonably stable.

An intramuscular injection in man caused moderate pain of short duration, and a slight local reaction lasting several days. This route could therefore be suitable where only a single dose would be given, as in prophylaxis or for treatment at a very early stage of infection. Trypanosomes disappeared within one hour of the injection.

The authors recommend that if mixtures of these particular substances be further examined the concentration of Mel B and novocaine should each be increased to 10 or 20 per cent., in order to minimize the amount of dimethylacetamide injected, since the latter is not free from toxicity.

iii. Since the advantages claimed for mixtures of Mel B and novocaine (see (i) above) are partly attributed to the fact that novocaine is a derivative of *p*-aminobenzoic acid, it was obviously desirable to study mixtures of Mel B and *p*-aminobenzoic acid itself.

Solutions of 3·6 to 5·6 per cent. *p*-aminobenzoic acid could easily be obtained in 3·6 per cent. Mel B in propylene glycol. Again it was shown that such

preparations consist of true mixtures, without chemical combination. They seemed fairly stable, though yellowish discoloration appeared more readily than in Mel B-novocaine mixtures.

In toxicity tests on two goats and a dog the intravenous injection of a dose containing 16 mgm. of Mel B per kgm. body weight was well tolerated, but intramuscular injections were so painful and caused such severe local reactions that this route of administration must be regarded as impracticable.

No tests have been carried out in man. *E. M. Lourie*

DEANE, Maria P. & DAMASCENO, R. M. G. Encontro do *Panstrongylus lignarius* naturalmente infectado por tripanosoma do tipo "cruzi" e algumas notas sôbre sua biologia. [*P. lignarius* **Naturally Infected by a Trypanosome of the "*cruzi*" Type, with Notes on its Biology.**] *Rev. Serviço Especial de Saûde Pûblica.* Rio de Janeiro. 1949, July, v. 2, No. 3, 809–14, 4 figs. on pl. English summary.

LEISHMANIASIS

FULTON, J. D., JOYNER, L. P. & CHANDLER, R. L. **Studies on Protozoa. Part II. The Golden Hamster** (*Cricetus auratus*) **and Cotton Rat** (*Sigmodon hispidus*) **as Experimental Hosts for** *Leishmania donovani*. *Trans. Roy. Soc. Trop. Med. & Hyg.* 1950, July, v. 44, No. 1, 105–12. [14 refs.]

The authors have previously shown that the cotton rat was highly susceptible to infection with *Leishmania donovani* [this *Bulletin*, 1948, v. 45, 775], but, unlike the golden hamster, in which the disease is fatal, the cotton rat does not suffer any ill-effects from the infection, which may last for more than one year. In the present paper the two animals are compared with regard to their suitability for chemotherapeutic tests. Drugs known for their leishmanicidal properties and also a number of new synthetic compounds were used in these tests. Preliminary toxicity tests on normal animals, carried out with stilbamidine, 2-hydroxystilbamidine, urea-stibamine, anthiomaline and sodium stilbogluconate, have shown that the two rodents differ in their tolerance to these drugs, but there were no marked differences in the therapeutic effect of the compounds tested. At any rate, none of those which were active in the hamster infection failed to affect the infection in the cotton rat. The results of the toxicity and therapeutic tests are fully recorded in tables, and the methods used are described in detail. It is concluded that the cotton rat is not only a suitable experimental host in leishmaniasis but is in some respects superior to the hamster. *C. A. Hoare*

GERMUTH, F. G., Jr., EAGLE, H. & OYAMA, V. **The Rate of Disappearance of** *Leishmania donovani* **from the Spleen of the Infected Golden Hamster following a Single Injection of a Pentavalent Antimonial.** *Amer. J. Trop. Med.* 1950, May, v. 30, No. 3, 371–6, 1 fig.

When an antimony compound is administered to a hamster infected with *Leishmania*, parasites gradually disappear from the spleen over a period of several weeks. In order to make a quantitative study of this phenomenon, the authors ground up weighed portions of spleen (excised 3 to 8 weeks after inoculation) with Locke's solution, prepared serial ten-fold dilutions, and incubated measured amounts of these dilutions with Locke-blood-agar medium

at 22°C. for 14 to 35 days. The reciprocal of the highest dilution which gave a positive culture was a measure of the number of viable organisms originally present per gm. spleen.

After a single injection of a pentavalent-antimony-sorbitol compound, viable parasites decreased in numbers for 16 to 32 days. It is suggested that the prolonged action may be due either to small quantities of antimony in the circulation, or to the presence of trivalent antimony deposits in the tissues.

The authors suggest that in human kala azar doses of antimony given once or twice a week should prove to be at least as effective as daily doses.

L. G. Goodwin

Germuth, F. G., Jr., Eagle, H. & Oyama, V. **An Evaluation of the Criteria of Cure in Experimental Leishmaniasis of the Golden Hamster.** *Amer. J. Trop. Med.* 1950, May, v. 30, No. 3, 377–85, 3 figs.

The authors have investigated a number of methods of detecting viable leishmanias in the tissues of infected hamsters after treatment with sub-curative doses of antimony. The most sensitive test was to expose the spleen through an abdominal incision, aspirate material with a 20-gauge needle and culture the material upon Locke-blood-agar or N.N.N. medium. Cultures made with small pieces of intact, or roughly crushed, spleen less frequently gave growth of the organisms present, and it was shown that the failure to grow was caused by the inhibitory action of the spleen tissue present.

Cultures made from liver gave fewer positives than did those from spleen, probably because of the smaller numbers of parasites present, or of their uneven distribution. Cultures grew most rapidly at 31°C., but also died out more rapidly ; it was concluded that the most favourable method for detecting the presence of scanty parasites was to incubate at 22°C. for 6 weeks.

It was estimated that a single negative result of a culture of aspirated spleen was " 80–85 per cent reliable " as a criterion of cure of the animal. This was confirmed by experiments in which normal hamsters were inoculated with splenic material from treated animals. In view of the prolonged action of antimony, it is recommended that tests of cure should not be performed until 6 weeks after the end of treatment.

L. G. Goodwin

FEVERS OF THE TYPHUS GROUP

v. Bormann, F. Zur Epidemiologie des Fleckfiebers. [**The Epidemiology of Typhus Fever.**] *Ztschr. f. Hyg. u. Infektionskr.* 1950, July 9, v. 131, Nos. 2/3, 169–97, 2 figs. [62 refs.]

The author describes a study of the epidemiology of louse-borne typhus fever, in 1942–1944, in the southern part of the Russian zone occupied by the Germans. The chief diagnostic method employed was a rapid modification of the Weil-Felix test.

The findings were in general agreement with those already reported by other German workers.

Among 1,527 healthy Russians living in areas of endemicity, but giving no history of previous attacks of typhus fever, positive reactions occurred in 24·5 per cent. Under the age of five about 11 per cent. were positive ; in the 6–10 age group 34·5 per cent. were positive ; between the ages of 11 to 50 the percentage of positives ranged between 21·3 and 29·5 per cent., and over the age of 50 it was 13·5 per cent.

The author believes that sub-clinical attacks played a greater part in causing positive reactions than undiagnosed ambulatory cases, and that the disease in the areas investigated was predominantly one of children.

Among 197 German soldiers who had spent more than a year in the endemic area without having had attacks of the disease the percentage of reactions was 46. Among 536 Germans of all ages living in parts of Germany where they had never been exposed to risk of infection 7 per cent. gave positive reactions. The significance of these "non-specific" reactions is discussed, their number suggests that the technique of the test may be suitable only for making a rough preliminary survey.

The incidence and severity of the disease were much lower among the persons with positive reactions than among those who were negative before the onset. The reaction was definitely positive in about 90 per cent. of patients in the second week of the attacks.

Partial immunity was believed to result in many cases from sub-clinical attacks and to be greatly increased by later attacks ; but even among persons with a high degree of immunity exposure to intense infection may break through the immunity.

No evidence could be found of the occurrence of racial immunity.

John W. D. Megaw

GIROUD, P. & WEI, W. P. Contribution à l'étude morphologique des corps homogènes constatés dans le poumon de lapin infecté de typhus épidémique. [**Morphological Study of the Homogeneous Bodies demonstrated in the Lungs of Rabbits infected with Exanthematic Typhus.**] *C. R. Soc. Biol.* 1950, June, v. 144, Nos. 11/12, 794–5.

The following is a translation of the authors' summary :—

It must be concluded that the homogeneous bodies have the same properties as the nuclei of bacteria or rickettsiae. This confirms our opinion on the development of rickettsiae and the nature of the homogeneous bodies.

H. J. O'D. Burke-Gaffney

SOMAN, D. W. **The Incidence and Distribution of Murine Typhus amongst Bombay Rats.** *Indian Med. Gaz.* 1950, June, v. 85, No. 6, 249–53. [13 refs.]

The incidence of murine typhus among the rats of Bombay City was investigated by the complement-fixation test in which the purified antigens employed were supplied by Dr. R. J. Huebner, Dr. R. R. Parker, and Dr. H. R. Cox. The Bengtson [here, as often, wrongly spelled Bengston] technique in a slightly modified form was adopted. In preliminary tests the Weil-Felix and rickettsia-agglutination tests were found less reliable.

Among 323 *Rattus rattus* 19·2 per cent. were positive ; among 193 *R. norvegicus* 45·0 per cent. were positive, and among 208 *Gunomys kok* 6·2 per cent. None of the 15 shrews, *Suncus caeruleus*, was positive.

In 34 attempts to isolate strains of rickettsiae from pools of brain suspensions, obtained from 119 rats, two strains of *Rickettsia mooseri* were isolated, one from *R. rattus* and one from *R. norvegicus*. [In the table the latter strain is shown as coming from *R. rattus* and the date of inoculation is given as July, 1947 instead of June, 1947 as stated in the text.]

With one of the strains a laboratory-bred white rat was inoculated ; 510 days later a guineapig inoculated with the rat's blood gave a febrile reaction, and a positive complement-fixation reaction at a titre of 1–20, but no scrotal reaction. Another guineapig, inoculated with the blood of a rat that had been

inoculated with a human strain of *R. mooseri* 522 days previously, responded with fever, a scrotal reaction, and a complement-fixation reaction at a titre of 1–5.

John W. D. Megaw

Reilly, W. A. & Earle, A. M. **Rocky Mountain Spotted Fever treated with Chloramphenicol and Aureomycin.** *J. Pediatrics.* St. Louis. 1950, Mar., v. 36, No. 3, 306–9.

"Six patients with rickettsial diseases with clinical findings and skin rashes typical of R.M.S.F. were successfully treated, four with Chloromycetin and two with aureomycin.

"One patient suffered a relapse, possibly due to too small a dose of the drug initially, but responded on reinstitution of aureomycin.

"The response to Chloromycetin in four patients and to aureomycin in one patient was in keeping with the findings of others."

Cutileiro, J. Considerações acerca do tratamento das rickettsioses exantemáticas pela aureomicina. [**The Treatment of Exanthematic Rickettsial Diseases with Aureomycin.**] *Gaz. Méd. Portuguesa.* 1950, v. 3, No. 2, 337–45, 1 chart. English summary.

Nine cases of tick-borne typhus fever of the Rocky Mountain spotted fever type and two cases of murine typhus were treated with aureomycin. In spite of considerable variations in the dosage and in the day of the disease on which treatment was started the results were uniformly very good. Some of the patients had nausea and vomiting; one patient suffering from murine typhus refused to continue the treatment after four days, starting from the 3rd day of the fever, the daily dosage was 1·4 gm. and the response was prompt, but there was a slight relapse on the 12th day; this was the only case in which a relapse occurred.

The results obtained with a daily dosage of 2·0 gm. on the first day and 1·0 gm. on the following days were as good as those observed in cases in which 6·0 gm. were given on the first day and 1·6 gm. on the following days. The author suggests that it would be quite justifiable to give 1·0 gm. daily for five or six days, reserving larger doses for cases in which there is danger to life or in which the smaller doses do not control the temperature in two or three days. The drug was given in divided doses every six hours, by the mouth. [No mention is made of the city in which the Hospital Curry Cabral is located. The journal is published in Portugal and there is no indication to show where the cases originated.]

John W. D. Megaw

Moeschlin, S. & Koszewski, B. J. Komplikationen des Q-Fever. [**Complications of Q Fever.**] *Schweiz. med. Woch.* 1950, Sept. 2, v. 80, No. 35, 929–31, 4 figs. [10 refs.]

In 9 out of 50 proved cases of Q fever the following "extra-pulmonary" complications were observed: (1) thrombophlebitis with pulmonary embolism in five cases with onset on the 15th to the 29th day and a duration of three to four weeks; (2) epididymitis in three cases, starting on the 27th to the 45th day, and lasting about one week; (3) one case of encephalitis occurred on the 18th day and lasted six weeks; (4) one case of pancreatitis occurred on the 24th day and lasted one week.

The author regards the complications as having been due to rickettsial activity; in one case the urine of the patient, taken five weeks after the onset, was inoculated into a guineapig which afterwards gave a positive complement-fixation reaction.

The relative frequency of these complications indicates that in the more severe attacks of the disease patients should be kept under observation for at least three or four weeks after complete defervescence.

In the only case in which two complications, thrombophlebitis and epididymitis, occurred in the same patient treatment with aureomycin had been given with apparent success ; in the case of pancreatitis both aureomycin and penicillin had been given.

The authors mention that among nine cases with early meningismus the cell count of the cerebrospinal fluid was increased in only one patient.

John W. D. Megaw

MEIKLEJOHN, G. & LENNETTE, E. H. **Q Fever in California. I. Observations on Vaccination of Human Beings.** *Amer. J. Hyg.* 1950, July, v. 52, No. 1, 54–64, 1 fig. [36 refs.]

Fifty members of the staff of the Viral and Rickettsial Diseases Laboratory of the California State Department of Health were vaccinated with one or other of two types of yolk-sac-ether-extracted Q fever vaccine. Among 40 persons who received three doses at weekly intervals 24 developed complement-fixation titres of 1–8 or more. Two of the three persons who received two doses had titres of 1–16, but only one of the seven who received a single dose had a titre of 1–8.

Eight of the persons who failed to give an antibody response to three doses of vaccine were again vaccinated eight weeks later, and six of them developed titres of 1–8 or more. The titres tended to fall considerably within four or five months and it was thought advisable to give "booster" doses every three months.

Severe local reactions sometimes occurred after "booster" doses of the more potent of the two vaccines ; five persons had large persistent tender swellings ; in seven cases fluctuation was detected and in four of these large abscesses were formed.

The fixation titre usually became much higher after "booster" doses, but a few persons gave a very sluggish response to repeated vaccination.

During the period of 16 months over which vaccination was continued no clinical case of Q fever occurred among the vaccinated, but unexplained rises of titre suggested that sub-clinical infection occurred in six persons. The only member of the staff who had not been vaccinated was attacked by Q fever three weeks after starting work in the laboratory. The patient responded promptly to aureomycin ; two doses of 2·0 gm. each were given with an interval of four hours, then 1·0 gm. was given every six hours for three days. There was troublesome nausea and vomiting.

John W. D. Megaw

BARTONELLOSIS

LASKOWSKI, L., PINKERTON, H. & GREIFF, D. **A Non-Pathogenic Bartonella-like Organism developing in Embryonate Eggs after Injection with** *Haemobartonella muris*. *J. Immunology*. 1950, June, v. 64, No. 6, 455–62, 4 figs. [16 refs.]

MAYER, M. La aureomicina en la anemia de las ratas causada por la *Bartonella muris* Mayer. (Nota preliminar). [**Preliminary Note on Aureomycin in Anaemia of Rats caused by** *B. muris*.] *Archivos Venezolanos de Patol. Trop. y Parasit. Méd.* 1949, Oct., v. 1, No. 2, 329–34.

The English summary appended to the paper is as follows :—

"Aureomycin in small single doses was very effective against Bartonella muris infection of white rats, when given in the first days after splenectomy

also when given one or three days before. Further experiments with Aureomycin and with other antibiotics will follow."

RABIES

REMLINGER, P. & BAILLY, J. Apparition "de novo" de certaines maladies infectieuses. Cas de la pseudo-rage. [*De Novo* **Appearance of certain Infectious Diseases: The Case of Pseudo-rabies.**] *Maroc Méd.* 1949, Sept., v. 28, No. 292, 485–8.

LIEOU, Y. C. & KOUO, C. C. Premier virus rabique "renforcé" isolé à Changhai. [**First "Reinforced" Strain of Rabies Virus Isolated in Shanghai.**] *Ann. Inst. Pasteur.* 1950, July, v. 79, No. 1, 108–9.

A street virus has been isolated from a dog which died after three days of illness without any clinical sign characteristic of rabies. On first inoculation into rabbits it produced rabies after six days. Fixed rabies virus vaccine gave good protection against this strain, and vaccine prepared from this strain gave good protection against the fixed rabies virus. When infected subcutaneously all dogs and half of the rabbits developed rabies. *C. R. Klimt*

KIRK, R., HASEEB, M. A. & DAVIS, A. T. **Attempts to cultivate Rabies in a Cell-Free Medium.** *J. Trop. Med. & Hyg.* 1950, Aug., v. 53, No. 8, 167–70.

The work described is yet another attempt at repeating VEERARAGHAVAN's work [this *Bulletin*, 1948, v. 45, 855], claiming multiplication of the rabies causative agent—which he believed to be a protozoon—in a cell-free culture medium. The authors, in common with previous workers in this field, failed to confirm the original work; indeed they demonstrated considerable loss of virus activity after cultivation. [The authors' own observations read as follows: "If anything there is a *slight* fall in the LD_{50} after cultivation".

Calculation from their data on the fourth experiment for example, however reveals an LD_{50} end-point prior to cultivation ranging between 10^{-5} and 10^{-6} and after cultivation of $10^{-3.6}$. This implies the loss of about two logs of titre, or approximately 99 per cent. of the original virus activity.]

No attempt at serial passage was made. [The lack of success in obtaining serial passage has always been the main argument against Veeraraghavan's claim.

Potency testing of a phenolized vaccine prepared from the culture virus to which reference has been made awaits a later report.

[The present work seems to have been hampered by the lack of adequate numbers of mice. Titrations carried out with 2, 4 and 5 animals per dilution respectively and not always covering the LD_{50} end-point dilution must be regarded as inadequate for statistical considerations.] *C. R. Klimt*

LIEOU, Y. C. & KOUO, C. C. Inoculation intracérébrale à travers l'orbite ou la fosse nasale chez la souris blanche. [**Intracerebral Inoculation through the Orbit or the Nasal Fossa in White Mice.**] *Ann. Inst. Pasteur.* 1950, July, v. 79, No. 1, 109–11.

The authors, who had learned of previous work in this field only after their own experiments had led to certain results, recommend trans-orbital and trans-nasal intracranial injections in mice. They fix the unanaesthetized mouse by clamping it in a tube of wood cut into two halves. For trans-orbital injection

they advance a fine needle from the inner angle of the orbit obliquely upwards for 0·8 centimetre and then deposit 0·05 of the inoculum. For the transnasal route they advance 1·5 centimetres up the nasal cavity and through the bottom of the cranium till they inject the same amount. Using these methods the authors claim 100 per cent. results in infecting mice with rabies or Aujeszky's Disease. *C. R. Klimt*

REMLINGER, P. & BAILLY, J. Action de l'auréomycine sur le virus rabique. [**Action of Aureomycin on Rabies Virus.**] *Bull. Acad. Nat. Méd.* 1950, v. 134, Nos. 21/22, 442–3. [Also in *Méd. Colonial.* Madrid. 1950, Aug. 1, v. 16, No. 2, 121–3.]

The authors have carried out three neutralization tests in rabbits and one post-infectious treatment test in dogs. The neutralization test was performed as follows :—

Tests 1 and 2 : One volume of 5 per cent. *virus fixe* suspension in physiological saline was treated for 5 hours at +6°C. with 1, 2, 3 and 4 volumes of a 0·25 per cent. aureomycin suspension (the authors complain that the aureomycin was not completely soluble).

In the first test, after subdural infection, the two controls died after 8 days, while of the 4 test animals 3 survived and one succumbed to rabies after 27 days. In the second test, which was in all respects identical with the first, only that *in vitro* contact with aureomycin was reduced to one hour, out of 4, 3 survived and one died of rabies on the 12th day.

The third test was carried out with 1 per cent. suspension of street virus and the injection was given subdurally. One out of 4 survived, the other three succumbed after 21, 27 and 33 days respectively. The two controls died on the 18th and the 20th days respectively.

The fourth test was carried out in dogs, infected by intramuscular injection with 5 per cent. street virus suspension. One was kept as control while the other received 50 mgm. of aureomycin hydrochloride orally every 6 hours. The control died of rabies on the 14th day, while the treated animal died on the 22nd day. The author further reports that he had had negative results on testing aureomycin against the virus of Aujeszky's Disease.

C. R. Klimt

BATES, J. D. **Failure of Antibiotics in Experimental Rabies.** [Correspondence.] *Nature.* 1950, July 22, 155–6.

The author, in Khartoum, records a small experiment in which 5 rabbits were inoculated intracerebrally with the strain of fixed rabies virus described by KIRK [this *Bulletin*, 1939, v. 36, 731] 24 hours after treatment had been started with one of three antibiotics, as follows :—

Rabbit 1.—Chloromycetin, 100 mgm./kgm. daily by mouth in 3 divided doses.

Rabbit 2.—Chloromycetin 200 mgm./kgm. as above.

Rabbits 3 and 4.—Penicillin in procaine oil, 20,000 u/mgm. daily.

Rabbit 5.—Streptomycin 40 mgm./kgm. daily intramuscularly in 2 divided doses.

All 5 rabbits died on the 5th day and no change in the incubation period or manner of death was observed.

The author concludes [though perhaps on limited experimental evidence] that " it would appear that the antibiotics used have no effects on the progress of experimentally induced rabies ". *H. J. O'D. Burke-Gaffney*

LIEOU, Y. C. & KOUO, C. C. Passages en série de virus de la maladie d'Aujeszky par scarifications de la peau chez le lapin. [**Serial Passage of the Virus of Aujeszky's Disease by Scarification of Skin in Rabbits.**] *C. R. Soc. Biol.* 1950, May, v. 144, Nos. 9/10, 629–30.

In 1947 the authors isolated in Shanghai a strain of the virus of Aujeszky's disease from the brain of a cat. Serial passage was achieved by infection of the scarified skin of rabbits with infected rabbit's spinal cord. Consequent on such passages the brain seemed almost to have lost its original infectivity, while the infectivity of the spinal cord remained unchanged ; moreover, as compared with the intracerebral route, the method of infection now described increased the period between infection and death from 1 to 3 days. *C. R. Klimt*

PLAGUE

HERBERT, D. **Studies on the Nutrition of *Pasteurella pestis*, and Factors affecting the Growth of Isolated Cells on an Agar Surface.** *Brit. J. Exper. Path.* 1949, Dec., v. 30, No. 6, 509–19, 1 fig. on pl. [16 refs.]

The author has confirmed the observations of previous workers who have found that *Pasteurella pestis* grows on ordinary agar when a heavy inoculum is applied to the plates, whereas single isolated cells do not grow unless blood or certain bacterial products are added to the culture medium, or unless anaerobic conditions are provided.

In a study of three virulent, and one avirulent, strains it was found that isolated colonies behaved as aerobes when grown on plain agar to which pure haemin was added. A reducing agent s ch as thiolacetate acted in the same way as haemin.

The author suggests that the failure of growth on ordinary agar is due to the formation of hydrogen peroxide under aerobic conditions and that reducing agents may neutralize the peroxide or may prevent its formation by rendering the conditions of growth sufficiently anaerobic. Haemin is assumed to act by promoting the synthesis of bacterial catalase. *John W. D. Megaw*

WON, W. D. **The Production of "Giant" Cells of *Pasteurella pestis* by Treatment with Camphor.** *J. Bacteriology.* 1950, July, v. 60, No. 1, 102–4, 1 fig.

Camphor has been found to produce development of "giant" cells in yeast (*Nature*, 1943, Nov. 6, 526). The present author has successfully demonstrated a similar production of such "giant" cells in cultures of *Pasteurella pestis* by incorporating camphor in the culture medium.

The *P. pestis* (virulent strain, Shasta) was grown on DOUDOROFF's liquid medium [this *Bulletin*, 1943, v. 40, 775]. Inocula were prepared to contain about 60 viable cells per ml. This suspension was used in 0·1 ml. quantities to inoculate blood agar plates incorporating 25 mgm. to 55 mgm. of crystalline camphor per 100 ml. of melted agar. After inoculation, the plates were incubated at 37°C. for 96 hours.

On control plates, colonies of *P. pestis* grew in 48 hours, but on the camphorated medium macroscopic colonial growth did not appear for 96 hours. There was no striking difference in the appearance of the colonies.

However, the cells from many colonies on the camphorated medium were obviously longer than those from control colonies and in some cases were twice as long and rather wider. The camphor-induced variants were stable

and did not revert nor dissociate after passage in animals and on medium. Increasing concentrations of camphor seemed to reduce the characteristic pleomorphism of *P. pestis*. [The microscopic appearances are shown in photomicrographs.]

Virulence of the " giant " and control cells was determined by intraperitoneal inoculation of mice : in the case of the " giant " cells, subculture was first made for a week on camphor-free medium. " Giant " cells proved to be as virulent as control cells. Quantitative virulence tests are being made with " giant " cells from different strains of *P. pestis*.

The author adds that the effective concentration of camphor necessary to induce the development of large numbers of " giant " cells of *P. pestis* does not correspond with the quantity added, owing to rapid volatilization at 37°C. It is suggested that rapid penetration of camphor into the cells may be an important factor in producing the " giant " variant.

H. J. O'D. Burke-Gaffney

CHOLERA

i. Collier, H. O. J. & Waterhouse, Pamela D. **Studies in the Chemotherapy of Cholera. II.** *In vitro* **Vibriostatic Properties of certain 2 : 4-Diaminopteridines.** *Ann. Trop. Med. & Parasit.* 1950, July, v. 44, No. 2, 156–60.

ii. ——, Hall, Iris F. & Waterhouse, Pamela D. **III. The Action of Pteridine-Sulphonamide Mixtures upon** *Vibrio cholerae* **and upon the Mouse.** *Ibid.* 161–7, 4 figs. [10 refs.]

i. In an earlier paper [see this *Bulletin*, 1950, v. 47, 236] Collier, Hall and Waterhouse had discussed methods for evaluating experimentally the action of drugs on *V. cholerae* in relation to selecting compounds which might suitably be tested for their therapeutic effect. The results are now reported of the *in vitro* activity of a number of new 2 : 4-diaminopteridines containing different substituents in the 6 : 7 position.

From initial suspensions containing 640 μgm. per ml. of each compound in peptone-water, serial dilutions were prepared and inoculated to a concentration of 10^3 vibrios per ml. After incubation at 37°C. the test cultures were examined at 16, 24 and 48 hours, and subsequently. On the basis of several repeat tests the minimal inhibitory concentrations were determined. The compounds found most active were subjected to further tests with an inoculation of 10^6 vibrios per ml. The results with 29 compounds and with sulphaguanidine in comparison are shown in a table.

Of the compounds tested the most active was 2 : 4-diamino-1′-methylindolo (2′ : 3′-6 : 7)-pteridine (0/120/II). Its inhibitory effect was more prolonged than that of other compounds and it was equally as active in peptone-water as in the synthetic medium of Sadusk and Oswald in which comparative tests were made. Another compound 2 : 4-diamino-6 : 7 di*iso*propylpteridine (0/129) which showed inhibitory action for a short period, and also sulphaguanidine, were more active in the synthetic medium than in peptone-water, which appears to contain antagonists to them. The compound 0/120/II differed from the others tested in being as active against an inoculum of 10^6 vibrios per ml. as against 10^3. As the free base of 0/120/II does not dissolve readily at 37°C. at or near neutrality, the phosphate, nitrate and chloride were prepared. The phosphate was found to be the most active of the salts and showed vibriostatic activity over the range of *p*H at which *V. cholerae* will grow. It was effective in much higher dilution than sulphaguanidine.

ii. From the series of 2 : 4 diaminopteridines dealt with in the preceding paper eight were selected for the study of their action in mixture with sulphaguanidine. The minimal vibriostatic concentration *in vitro* was ascertained for each pteridine, sulphaguanidine and a series of mixtures of the two. Intraperitoneal toxicity for mice was also determined in the case of three of the pteridines and their admixtures with sulphaguanidine.

All the pteridines were found to synergize powerfully with sulphaguanidine in peptone-water, a mixture of 10 per cent. pteridine being about equal to pure pteridine over a period of 24 hours' incubation. At longer periods of incubation the mixture was more active than pure pteridine. The *in vitro* potentiation of sulphaguanidine was less when the test was carried out in the synthetic medium of Sadusk and Oswald. While the two pteridines 0/129 and 0/120/II were more active against *V. cholerae* than sulphaguanidine, they were also more toxic to mice. Mixtures with sulphaguanidine, however, were less toxic than pure pteridine and may be even less toxic than sulphaguanidine alone. The findings are considered to suggest that the addition of a small amount of an active pteridine might increase the value of the sulphonamide for the treatment of cholera, without seriously increasing toxicity. The compounds 6 : 7-di*iso*propyl and 6 : 7-di-(p-methoxyphenyl)-pteridine antagonized sulphaguanidine to some extent in intraperitoneal toxicity to mice.

J. Taylor

AMOEBIASIS AND INTESTINAL PROTOZOAL INFECTIONS

Neghme, A. & Silva, R. Epidemiología de las parasitosis intestinales en el medio rural de Chile. I Comunicación. [**Intestinal Parasitism in Rural Districts of Chile.**] *Rev. Chilena Hig. y Med. Preventiva.* 1949, Mar., v. 11, No. 1, 17–23. English summary (5 lines). II Comunicación. Primeros resultados del censo enteroparasitario en las provincias centrales. *Ibid.*, 25–35. English summary.

This article is issued in two parts. In the first, the general sanitary, or rather insanitary, condition of the rural areas of Chile is considered ; in the second, the results of a limited number of faecal examinations are reported. In December, 1948, a fire at the School of Medicine destroyed the records of 5 years' work ; hence the data presented are unavoidably incomplete.

In the first part, the general hygiene of parts of Santiago, Aconcagua, Osorno and Concepción provinces are detailed and, hygienically speaking, they are in a poor way. The drinking water comes largely from trenches or canals and irrigation water, not only liable, but actually subject, to excretal pollution ; the people live in rather crowded communities and malnutrition and alcoholism are common. The districts were divided into sectors and in each sector endeavour was made to obtain a cross-section of the people and some 15 to 20 per cent. of them were examined. At first only one faecal specimen of each was examined but it was found that nearly one-third was missed and the single examination was replaced by three ; one on a slide direct, one stained with Lugol's iodine, and one by Telemann's enrichment method. The authors have in view other methods for preservation of specimens for more thorough examination.

The results are detailed in the second communication and 580 are here analysed in 5 rural zones, Pirque, El Volcán, Maipo, Melipilla and San Roque. The numbers and percentages for each of these are given as regards dysentery amoebae, *Entamoeba coli*, *Endolimax nana*, *Giardia intestinalis*, *Hymenolepis nana*, *Ascaris lumbricoides*, *Trichuris trichiura* and *Enterobius vermicularis*.

Amoebiasis involves a much higher percentage in rural than in urban districts, in fact, three or four times as high and as much as 36 to 40 per cent. ; *E. coli* is as high as 80 per cent. and *Endolimax nana* 11·2 per cent. in the Pirque district. Giardiasis has an incidence up to 29·5 per cent. in El Volcán and generally is about double the urban rate of infection. [The rates here and in several places of this article are liable to mislead, being based on very small totals ; thus *Giardia* specimens total only 80 in all five districts together, and *E. nana* 46.] Ascariasis was found in 10·2 per cent. (only 8 positive) in San Roque and 24·3 per cent. in Melipilla and 24·2 per cent. in Maipo. Of *Enterobius vermicularis* the authors state that of 332 persons examined there were marked variations from district to district, but that in the case of this parasite alone was the rural infection rate below the urban [but no figures of incidence are given]. The conditions in rural areas are undoubtedly bad and could be much improved by better sanitation and more education. *H. Harold Scott*

Hewitt, R., Wallace, W. & White, Elizabeth. **The Effect of Aureomycin on *Endamoeba histolytica* in Vitro.** *Science.* 1950, Aug. 4, 144–6.

The authors describe the results of *in vitro* tests of aureomycin hydrochloride on *Entamoeba histolytica*, in which they used cultures with a mixed bacterial flora, grown in a medium consisting of liver infusion agar (Difco) slope overlayed with Loeffler-Ringer solution or horse-serum-saline and containing rice starch. Tests were carried out by two methods. In one, varying dilutions of the drug were dissolved in the overlay, after which the amoebae were inoculated into the medium, 0·1 ml. of a rich 48-hour culture being used. At the end of 48 hours at 37°C. the cultures were examined microscopically. Growth of the amoebae was scarce or completely inhibited in dilutions of aureomycin up to 1 : 100,000, but since bacterial growth was also considerably inhibited at high dilutions of the antibiotic and completely prevented at low dilutions, it could not be determined by this method whether aureomycin had a direct effect upon the amoebae.

This element of doubt was excluded by using the second method, in which 2 ml. amounts of pooled overlays of 48-hour cultures of amoebae were cultivated in 8 ml. of saline, to which aureomycin was added. After exposure from 10 to 60 minutes, the mixture was repeatedly centrifuged at 2,000 r.p.m. for 3 minutes, decanted and washed with saline. The sediment containing amoebae was then inoculated in media previously seeded with the original bacterial flora, and the resulting growth of amoebae was recorded after 48 hours' incubation. It was shown that aureomycin had a marked amoebicidal effect in a dilution of 1 : 1,000 and less pronounced effects at 1 : 10,000. In comparison, emetine showed less amoebicidal action after contact for 60 minutes at dilutions of 1 : 1,000. It was found that there was no measurable difference in the effect of these dilutions of aureomycin at *p*H varying between 3·12 and 8·00. Similar *in vitro* tests were made with 1 : 1,000 dilutions of streptomycin, bacitracin and chloramphenicol, but none of these antibiotics had any measurable effect on *E. histolytica* after exposure for one hour.

It is concluded that, although it cannot be inferred from these *in vitro* tests that aureomycin has a similar effect in human infections, this possibility must be taken into consideration. [See also Jones, this *Bulletin*, 1950, v. 47, 748.] *C. A. Hoare*

Bose, A. N., Bose, S. & Ghosh, T. N. **Studies on Arsenoxide Derivatives. Part I. Toxicity of Carbamidophenylarsenious Acid in Comparison with Carbarsone.** *Indian Med. Gaz.* 1950, Feb., v. 85, No. 2, 50–54, 2 figs.

Carbarsone is a pentavalent arsenical, the trivalent analogue of which is *p*-carbamidophenylarsenious oxide. Anderson *et al.* [this *Bulletin*, 1948,

v. 45, 520] claimed that the latter is the more effective compound against amoebiasis, and the present authors have accordingly prepared their own supply, in the hydrated form (*p*-carbamidophenylarsenious acid, or " C.P.A."), and studied its toxicity in mice in comparison with that of carbarsone.

The tests consisted of dose-mortality observations after oral and subcutaneous administration of large doses (for acute toxicity), and of smaller and repeated doses (for chronic toxicity). Chronic toxicity was also studied by weighing the mice during and after a course of daily treatment. Histological observations were made on poisoned animals, and the organs mainly affected were shown to be the liver and kidneys. Estimations of blood concentration after oral administration of about half the well-tolerated dose showed that the drug is fairly well absorbed from the gastro-intestinal tract, and disappears from the circulation in about 5 hours.

" C.P.A." proved to be more toxic than carbarsone.

[It is, of course, a well-known general rule that trivalent arsenicals are more toxic (though not necessarily with a less favourable chemotherapeutic index) than their pentavalent analogues.] *E. M. Lourie*

Pérez Santiago, E. & Hernández Morales, F. **The Treatment of Amoebiasis with Milibis (Bismuthoxy-para-N-glycolylarsanilate). Preliminary Report.** *Puerto Rico J. Pub. Health & Trop. Med.* 1950, June, v. 25, No. 4, 419–22. [Spanish version 423–7.]

" Milibis ", bismuthoxy-para-N-glycolylarsanilate, was made in Germany by I. G. Farben and first used in 1942 under the name of " Wia ". It is a flesh-coloured powder, insoluble in water, and contains 15·7 per cent. of arsenic in the pentavalent form and 37 per cent. of bismuth. The presence of the bismuth lowers the solubility of the arsenic and thus reduces its absorption from the intestine ; hence the low toxicity of the drug.

Milibis *in vitro* kills amoebae within 24 hours in dilutions of 1/1,000 and 1/2,500. Hauer [this *Bulletin*, 1943, v. 40, 913] found it therapeutically effective in acute amoebiasis in cats in doses of 30 mgm./kgm. of body weight when given once or twice daily ; and he showed that the tolerated single dose in rabbits was 2 gm./kgm. and in mice 100 mgm./kgm. of body weight. Others have found Milibis to be as effective an amoebicide as carbarsone and less toxic [this *Bulletin*, 1950, v. 47, 1088].

Twenty-two patients, 20 of them passing cysts and 2 trophozoites of *Entamoeba histolytica*, were treated with Milibis. Two tablets (500 mgm.) were given orally thrice daily for 7 days ; the patients' ages ranged from 10 years to over 40 years ; no serious toxic signs were observed, but 3 patients had slight nausea and epigastric discomfort, one suffered from dizziness and anorexia, and another from slight lower abdominal pain. Twenty (91 per cent.) of the patients were apparently cured of their amoebic infections (an average of 10 negative stools from each was examined). Repetition of the treatment four months later in one of the original failures to cure produced the desired result.

The authors concluded that Milibis seems to be effective in the treatment of uncomplicated lumen infections of *E. histolytica*, and that it is well tolerated.

A. R. D. Adams

Basnuevo, J. G. & Gutiérrez Estarlí, E. Siete casos de hepatitis tratados con cloroquina (Tanakán). [**Seven Cases of Hepatitis treated with Chloroquine (Tanakan).**] *Rev. Kuba Med. Trop. y Parasit.* 1950, Mar.–Apr., v. 6, Nos. 3/4, 45–54, 3 figs. & 1 diagram. [35 refs.] English summary.

Basnuevo, J. G., Guerra Valdés, R., Gutiérrez Estarlí, E., Sánchez Beltran, O. Absceso hepático amibiano complicado (pulmón) curado con Tanakán (Cloroquina). [**Amoebic Complicated Liver Abscess cured with Tanakán Chloroquine**]. *Rev. Kuba Med. Trop. y Parasit.* 1950, Mar.–Apr., v. 16, Nos. 3/4, 33–9, 5 figs. [11 refs.] English summary.

Basnuevo, J. G. & Vázquez Rosales, G. Absceso hepatico amibiano perforado curado con emetina-cloroquina. [**Perforated Amoebic Liver Abscess cured with Emetine-Chloroquine.**] *Rev. Kuba Med. Trop. y Parasit.* 1950, Mar.–Apr., v. 6, Nos. 3/4, 54–7, 2 figs. [10 refs.] English summary.

Most, H., Miller, J. W. & Grossman, E. J. **Treatment of Amebiasis with Bacitracin and other Antibiotics.** *Amer. J. Trop. Med.* 1950, July, v. 30, No. 4, 491–7.

Treatment with the sulphonamides and penicillin has repeatedly been shown to influence the clinical course of amoebic dysentery. It is assumed that this is the result of modification of the intestinal flora, some of which apparently are necessary to the welfare of *Entamoeba histolytica*. Addition of the antibiotic bacitracin to cultures has now been found to inhibit growth of *E. histolytica*, possibly by the same means, and the administration of this compound to patients with amoebic colitis has resulted in the disappearance of the parasites from their stools, and clinical recovery.

Fifty-one proven cases of *E. histolytica* infection (13 asymptomatic ; 30 mildly symptomatic ; and 8 moderately or seriously ill) were treated orally with tablets of bacitracin ; the daily dosage, which varied from 40,000 to 120,000 units, was continued over from 5 to 20 days. In three cases only did parasites fail to vanish from the stools during treatment, but in 14 they reappeared in them in from 6 to 350 days thereafter (all but 4 of these parasitic relapses took place within 35 days). Nine of these patients were re-treated with bacitracin, but 6 of these again were not freed of parasites, and after a third course of treatment only 1 of the 3 receiving it was cured of the infection. Thus of 51 patients with amoebiasis treated for the first time with bacitracin 34 (66 per cent.) were freed of amoebae ; repetition of the treatment ensured sterilization in only 33 per cent. of the failures. Continuation of bacitracin treatment beyond a period of 10 days, or increase of the daily dosage beyond 80,000 units, did not significantly increase the probability of cure beyond 66 per cent.

In all of the 8 patients with moderate or severe clinical manifestations and sigmoidoscopic evidence of mucosal lesions there was striking clinical improvement and healing of the visible lesions within 4 to 14 days. One of these patients, an Indian having very severe amoebic dysentery with necrotic and gangrenous foci in the rectum, was given 50,000 units of bacitracin daily by mouth in divided doses and there was remarkable clinical improvement within the first 24 hours ; by the fourth day the rectal mucosa was almost normal in appearance. In another case, where emetine, the iodoquinolines and carbarsone had failed to arrest the acute attack, 80,000 units of bacitracin daily for 13 days produced an equally satisfactory response ; when relapse took place in this case 9 months later chloroquine, diodoquin, carbarsone, wia [" Milibis "] chloromycetin and aureomycin all failed to terminate the attack but bacitracin in daily doses of 160,000 units again produced remission. Bacitracin may thus prove effective in cases of amoebiasis when other therapeutic agents prove unavailing.

Toxic side-effects observed in 100 patients treated with bacitracin were negligible ; they took the form of distension and diarrhoea for one day in a single case, and some diarrhoea for 8 days in one other case. The drug is

apparently little, if at all, absorbed as none was demonstrable in the blood or urine of 5 patients 2 to 4 hours after single doses of 10,000 to 50,000 units of bacitracin.

The action of aureomycin on 8 cases of amoebiasis has already been reported by Most [*Bull. New York Acad. Med.*, 1949, v. 25, 717] who has now used the drug in 33 cases, 32 asymptomatic or mildly symptomatic and one with severe symptoms. The latter patient was given 23·25 gm. of aureomycin in divided doses over a period of 9 days without parasitological or clinical benefit, but in all the former 32 cases parasites vanished from the stools during the treatment (1 to 2 gm. daily for 10 days). In one of these 32 cases there was a parasitic relapse 15 days after completion of the treatment ; but none of the others have passed parasites over observation periods ranging from 2 weeks to a year, so the apparent cure rate of aureomycin treatment in amoebiasis is over 90 per cent. The toxic side-effects of aureomycin treatment were vomiting and diarrhoea, which occurred in about 20 per cent. of patients ; in some these were so severe as to necessitate the suspension of treatment after the 3rd or 4th days—before the parasites vanished. The more recent issues of the drug are less toxic than the earlier, and it is considered that 1 gm. daily is as therapeutically effective as 2 gm. daily.

Chloromycetin (1 gm. daily for 7 days) failed to influence the course of a severe attack of amoebic dysentery, and in doses of 3 gm. daily for 7 or 8 days it also failed to sterilize five asymptomatic carriers of their *E. histolytica* infections.

The authors conclude that bacitracin is effective in terminating acute attacks of amoebic dysentery and ensures parasitic sterilization of the bowel in two-thirds of cases of amoebiasis. Its use is associated with the disappearance of clostridia and enterococci from the stools, and this may afford an explanation of its action on the amoebae. Aureomycin, on the available smaller figures, seems to ensure a higher percentage of parasitic cures, and its use also is associated with the disappearance of faecal clostridia and a reduction in the number of enterococci in the stools.

[This paper, which is commendably restrained in its claims, affords valuable and concise information on the efficacy of the newer antibiotics in the treatment of amoebiasis. The criticism may be levelled that the number of stools examined in many cases regarded as presumptively cured is small (6–12).]

A. R. D. Adams

Gutch, C. F. **Aureomycin in the Treatment of Amebiasis.** *New England J. of Med.* 1950, Aug. 3, v. 243, No. 5, 185–6.

Twenty ex-service patients, with proven amoebiasis but with no lesions demonstrable sigmoidoscopically, were treated with 0·75 to 1·0 gm. of aureomycin every 6 hours by mouth to totals of 8 gm. to 28 gm. Side effects, which consisted of nausea, giddiness and headache, were minimal ; pruritus ani developed in two patients 4 to 6 weeks after treatment. Stool examination of all the patients was negative on completion of the treatment ; from 15 patients no parasites had been recovered up to 3 months or more after it, but from 2 patients parasites were recovered within six weeks ; one of these relapsed again after a second course of treatment while the other did not.

Aureomycin produces a cure rate comparable to that of other amoebicidal treatments, and it has the advantage of being well tolerated.

A. R. D. Adams

Diez Melchor, F. Disenteria balantidiana. [**Balantidial Dysentery.**] *Med. Colonial.* Madrid. 1950, Sept. 1, v. 16, No. 3, 179–89.

A general review.

v. Vietinghoff-Scheel, O. F. Atebrin zur Behandlung der Kaninchenkokzidiose Ziehl-Neelsen-Färbung von Leberkokzidien. [**Atebrin (Mepacrine) in the Treatment of Coccidiosis in Rabbits. Ziehl-Neelsen Staining of Coccidia in the Liver.**] *Arch. f. Hyg. u. Bakt.* 1950, v. 133, No. 1, 59–62. [12 refs.]

" The English summary appended to the paper is as follows :—

" In sections of rabbit liver, coccidia oöcysts can be made distinctly visible by means of the Ziehl-Neelsen staining method. In four rabbits suffering from coccidiosis, the disease and the oöcysts excreted in the feces were successfully treated with atebrin. The atebrin was dissolved in water and sprayed deeply into the gullet."

YAWS

Guimarães, F. N. Experiências com altas doses de penicilina-procainica no tratamento da bouba (framboesia, pian, yaws). [**Procaine Penicillin in Large Doses in the Treatment of Yaws.**] *Hospital.* Rio de Janeiro. 1950, Mar., v. 37, No. 3, 361–9. [33 refs.] English summary.

Procaine penicillin was chosen because it is slowly absorbed and therefore injections could be made less frequently. The 30 cases selected were in patients with primary and secondary yaws whose infection had existed for 2 months to 3 years. They were divided by age into 3 groups : those from 2 to 9 years receiving a total of 900,000 units in three equal doses ; those of 10 to 17 years 1,800,000 units ; those over 18 years 2,700,000 units, each in three doses, and injections were given on alternate days for 5 days. The results observed were as follows : bacteriologically, after 6 hours 19 still showed spirochaetes, 11 did not ; after 12 hours the figures were 9 and 21 respectively ; after 24 hours all were negative. Clinically, the lesions were retrogressing by the third or fourth day and cicatrization was complete in 10 to 15 days. Two who had also a fuso-spirillar infection [presumably tropical ulcer] cicatrized after 20 days. Two adults relapsed ; in one the initial lesion again broke down ; the other had relapsed previously, 2 years before, with plantar lesions. Serologically, 18 of the 30 were negative after treatment ; two early cases were negative before treatment was started.

The advantages of a reduction in the number of injections and the length of treatment are obvious and particularly useful in rural campaigns against the disease. *H. Harold Scott*

Ferreira, F. S. da C. & Pinto, A. R. O tratamento das boubas com o S.T.B. [**S.T.B. in the Treatment of Yaws.**] *Anais Inst. Med. Trop.* Lisbon. 1949, Dec., v. 6, 115–26, 8 figs. on 4 pls. English summary.

S.T.B. [this *Bulletin*, 1949, v. 46, 839] is a trivalent arsenical with the composition 4-oxy-3-acetylamine phenyl arsenoxide. It is given by mouth in doses of 0·01–0·02 gm. per kilo bodyweight, daily for 5 consecutive days, in tablets of 0·1 gm., the maximum dose being 0·5 gm.

Thirty-five yaws patients in Portuguese Guinea were thus treated, 17 males, 18 females, of whom 23 were children, 6 were adolescents and 6 were adults ; four were in the primary stage, 30 in the secondary with papules, maculae and ulcers, and one with extensive ulceration and a circle of satellite lesions. All the patients bore the drug well, showing no signs of intolerance. The effects

were certainly remarkable. At the end of the second day's treatment "70 per cent. of the lesions showed a noticeable diminution of exudation and many had disappeared entirely"; the next day crusts were forming, but 3 patients had increased exudation which also was purulent, possibly the result of secondary infection. By the end of the fourth day all the lesions were dry and some were cicatrizing. By the fifth day "37 per cent. of the cases" [14 patients ?] were clinically cured, with cicatrization leaving dark-coloured areas. Progress thereafter was on the whole good and when the patients were seen a month later, 31 were "clinically cured," two had relapsed, one still had suppurating lesions and one an uncured ulcer. After 3 months all were clinically cured, even those who had previously relapsed (these had been treated with stovarsol) and after 6 months the cure had been maintained; there had been no relapses. The Kahn test was carried out on 30 of the patients at the end of this period and 23 reacted positively. Photographs show the conditions before and after treatment in 4 of the cases [but how long after is not mentioned].

H. Harold Scott

HELMINTHIASIS

Weise, H. Ueber die Wurmbekämpfung durch Enzyme. [**Enzyme in the Treatment of Helminthiasis.**] *Med. Klin.* 1950, Sept. 1, v. 45, No. 35, 1096–8, 1 fig.

The author remarks, as many others have done, on the fact that helminthic infestation has increased in Germany since the war and in the southern districts more than the northern. After referring to the many methods and drugs which have been tried, he stresses the value of a papain preparation, made from *Carica papaya* and known as *Vermizyme*. This is given in dragées, 4 in number and followed by the same dose hourly for 4 hours, or 20 dragées in all for adults. In severe infestations 5 may be given for a dose, a total of 25. Its action is on the cuticle of the worm, *Enterobius* and *Ascaris* in particular, softening and disintegrating it so that the digestive juices act on the exposed body of the worm and destroy it. The drug is followed by a dose of castor oil; a few worms are expelled which were in the lower rectum; but examination later of the motions reveals none as they have been disintegrated in the bowel. An illustration shows the *in vitro* breaking up of *Ascaris suis* by action of Vermizyme solution.

H. Harold Scott

Watson, J. M., Zahawi, S., Naji, A. F. & Murtadha, M. **Studies on Bilharziasis in Iraq. Part III. Pulmonary Bilharziasis.** *J. Faculty of Med. Baghdad, Iraq.* 1949, Nov., v. 13, No. 6, 154–71. [34 refs.]

Pulmonary complications of Schistosomiasis are commonly reported, particularly in association with *Schistosoma mansoni* infections. These may be acute and early when many cercariae are passing through the lungs, or they may be chronic and occur later when ova and adult worms lodge there. In Iraq the latter chronic type has been noted from time to time at autopsy; in the present paper four such cases, two certainly due to *S. haematobium* infections [and the other two presumably so], are recorded in detail and discussed in the light of previous reports in the literature. In one of these four cases there was an associated tuberculous pulmonary infection, and the possibility was considered that the pulmonary bilharziasis predisposed to this. Examination of 109 unselected cases of pulmonary tuberculosis for schistosomal infections showed 13 of them to be passing bilharzia [? *S. haematobium*] ova

in the urine ; in two only of these were there indications of a lung bilharzia infection. There was no evidence of any direct association between the two lung infections. *A. R. D. Adams*

HALAWANI, A. & DAWOOD, M. M. **Recent Advances in the Treatment of Bilharziasis. Parts I and II.** *J. Roy. Egyptian Med. Ass.* 1950, v. 33, Nos. 2 & 5, 171–80 ; 463–80. [27 refs.]

I. The usual course of Fouadin treatment, or one of tartar emetic, entails the patient's attendance for 3 or 4 weeks ; a result is that many do not complete the course. Attempts have been made to abbreviate the treatments ; the authors summarize the methods so far advocated in the literature to this end and assess their merits and defects ; the latter they consider to outweigh the former. Recently they have treated 1,000 ambulant cases of schistosomiasis (960 *Schistosoma haematobium*, and 40 *S. mansoni* infections) with daily intramuscular injections of Fouadin (Repodral) on 10 of 11 consecutive days ; the daily dose for an adult was 5 cc. of a 6·3 per cent. solution, freshly prepared. The treatment was well tolerated and specimens from all but 154 of the *S. haematobium* and two *S. mansoni* cases so treated, contained no viable eggs the day after completion of treatment. On re-examination of 97 of the 154 *haematobium* and some [number unstated] of the *mansoni* cases from 2 to 4 weeks later, 85 of them were found to be negative ; thus though the apparent failure at the end of treatment was 7·3 per cent., the figure may in reality be much lower ; so this method of treatment, in comparison with others, proved very successful. Failures to cure were more frequent in children and adolescents than in adults, explanations being the smaller dosage of drug and possibly the more efficient excretion of antimony by the youthful kidney.

Shorter and more intensive courses of Fouadin treatment [?intramuscularly] were also tried. Three or four injections, each of 5 cc., at intervals of 3 hours failed to cure [? species] ; the urines of two patients given 24 cc. in one day were freed of ova, but there was excessive vomiting ; three injections each of 5·75 cc. on each of two consecutive days caused disappearance of the eggs [? species] but produced excessive side-effects.

Fifty patients given a four-day course of 5 cc. twice daily at intervals of 3 hours (total 40 cc.) passed no ova [? species] up to three weeks after its completion ; these patients were ambulant, and apart from vomiting the side-effects were not serious and they were lessened by giving hypertonic glucose intravenously.

Potassium antimony tartrate in 6 per cent. solution (1 grain in each cc.) was also used. The bulk of each injection was brought to 10 cc. by the addition of 5 per cent. glucose, and the dose of drug at each injection for an adult was 2 grains, given intravenously. The courses and their effect are summarized on page 1205 :—

Egyptian patients, owing to the intensity of their schistosomal infections, poor nutrition and physical defects, do not tolerate well two-day courses of Fouadin or emetic treatment. Patients in Egypt under treatment for bilharziasis can be classified into (1) those who are physically fit and whose pulse rate returns to normal within 2 minutes of an exercise test, (2) those who are fit but whose pulse rate returns to normal 2–3 minutes after an exercise test, (3) those with valvular cardiac lesions but good compensation and normal kidney function, and (4) those for whom antimony therapy is contraindicated on account of nephritis, heart failure, jaundice or fever. Class (1) may be given a four-day Fouadin, or a five- or ten-day emetic course, Class (2) a ten-day Fouadin course, and Class (3), which also includes pregnant women, the classical protracted alternate-day Fouadin course.

Course	Number of patients	Number freed of ova over 3 weeks	Side-effects
2 grains × 3 daily for 2 days ...	5	5	Severe
2 grains × 3 on 1st day × 2 on 2nd day	13	13	Severe
2 grains × 4 for 1 day	4	3	Severe
2 grains × 3 for 1 day	1	1*	Severe
2 grains × 2 daily for 3 days ...	15	13	Moderate
1·5 grains × 2 daily for 5 days (without glucose solution)† ...	50	50	Mild
2 grains × 1 daily for 10 days (without glucose solution)† ...	12	12	Mild

*Subsequently relapsed.
†Suitable for ambulant patients.

II. The authors review the available data on the results of treatment of bilharzial infections of mice, gerbils and monkeys with the thioxanthone derivatives, known as Miracils A, B, C and D, given orally or intraperitoneally; of these Miracil D has proved the most effective against both *Schistosoma haematobium* and *S. mansoni*, though in the only trial cited it was ineffective against *S. japonicum*. In experimental animals sub-lethal doses of the Miracils cause a fall in blood pressure and increased respiration ; their lethal action is due to direct action on the heart. In human beings therapeutic doses cause nausea, vomiting, insomnia, fine tremors, giddiness and a yellow coloration of the skin ; these are most pronounced during the first three days of treatment. Miracil D is less toxic than is Miracil B, and its side-effects can be lessened by giving glucose intravenously. Miracil D was first tried out in the field about the same time in Rhodesia and in Egypt. In Rhodesia doses of 100 to 300 mgm. daily for 6 days a week over a period of three weeks were employed ; though these were considered the maximum tolerated doses the results were poor [this *Bulletin*, 1948, v. 45, 526]. In Egypt doses of not less than 18 to 20 mgm. per kgm. daily for 7 to 8 days were found to be necessary to produce a temporary effect, but only 2 of 20 patients so treated after an interval failed again to pass living eggs in the urine [this *Bulletin*, 1949, v. 46, 753].

Recently the authors have treated over 150 cases of *S. haematobium* infection with Miracil D ; they have given doses of Miracil D ranging from 15 to 20 mgm. per kgm. of body weight daily for 5 to 9 days to boys with heavy *S. haematobium* infections in a reformatory school, and this course was repeated after a month's interval ; the cure rate was " not significant ". In no case of this series did Miracil D free the urine even temporarily of eggs when given in a dosage of less than 15 mgm. per kgm. for 5 days ; in children weighing from 30 to 38 kgm. doses of 16 to 20 mgm. per kgm., although producing reduction in the number of eggs excreted and freedom from haematuria, failed to clear the urine entirely of eggs. In light infections in children weighing from 27 to 40 kgm. 22 mgm. per kgm. for 5 days caused disappearance of ova from the urine by the third week in 3 of 8 patients ; but a nine-day course of 22 mgm. per kgm. daily freed 6 of 10 boys, each weighing 36 or 37 kgm., of ova after 3 weeks,

and 4 of these were still free from them after 3 months. That the results of treatment depend firstly on the age and weight of the patient and secondly on the intensity of the infection is shown by the treatment of 13 children weighing from 25 to 32 kgm. with 22 to 25 mgm. per kgm. of the drug daily for 10 days. Six of these children had very heavy infections ; 3 of them were free of eggs after one month, and only one of them after 3 months. Of 25 older children weighing 41–57 kgm. similarly treated, 18 were free of eggs 3 weeks after treatment and 13 after 3 months. The best results with Miracil D were obtained when 22 mgm. per kgm. were given to 20 patients as a single dose daily for12 days ; 19 were free of eggs after 2 weeks, and 16 after 3 months. [The results of the different dosages are set out in tables.]

Observation shows that the higher the incidence of schistosomiasis in a community the greater is the intensity of the infection to be expected in the individuals composing it. With increasing age the intensity of the infection in the individual decreases, possibly as a result of increasing immunity or, more probably, as a result of lessened exposure to infection, as young children tend to play in water. The younger the affected person the greater is the daily dosage of drug required and the more prolonged is the course necessary to ensure cure, in view both of the heavier infection and also of the more efficient metabolism and excretion of the drug by the youthful.

The chief side-effects of Miracil treatment have already been noted ; in addition to these haematemesis occurred in two cases, and angio-neurotic oedema of the face in two others. Extreme weakness and inability to get up, to an extent such as to resemble myasthenia, were also seen in one of 142 cases treated with Miracil D, and in one of 5 treated with Miracil B ; liver tenderness, in conjunction with extreme weakness and with tremor of the fingers, was observed in two cases of *S. mansoni* splenomegaly treated with Miracil D.

A. R. D. Adams

HASKINS, W. T. & LUTTERMOSER, G. W. **The Comparative Toxicities of the Antimonyl Derivatives of the Four Isomeric Potassium Acid Tartrates.** *Amer. J. Trop. Med.* 1950, July, v. 30, No. 4, 591–2.

"The results are summarized in the table. No significant difference in toxicity was found among the four isomers."

RODRIGUEZ-MOLINA, R., ACEVEDO, C. E., TORRES, J. M., LOPEZ-SANABRIA, U. & RAMIREZ-RODRIGUEZ, E., with the technical assistance of R. DIAZ-CALDERIN. **Rectal Biopsy as a Criterion for Evaluation of Treatment in Schistosomiasis mansoni.** *Bol. Asoc. Méd. de Puerto Rico.* 1950, July, v. 42, No. 7, 415–18.

Twenty-nine adult male Porto Ricans were treated for *Schistosoma mansoni* infections with 3 cc. of anthiomaline given intramuscularly every 48 hours for 10 injections. These patients had only mild or moderately severe chronic infections with no clinical evidence of visceral disease ; the diagnosis had been made in many of them by the fortuitous discovery of ova in the stools.

Rectal biopsies, first described by OTTOLINA and ATENCIO [this *Bulletin*, 1944, v. 41, 945] and subsequently modified by HERNÁNDEZ-MORALES and MALDONADO [*ibid.*, 1947, v. 44, 330], were performed by taking material from the first or the second rectal fold with a Hajek sphenoid punch ; this was then crushed between two slides and searched microscopically. On completion of the treatment, ova were recovered by this means from 15 (51·6 per cent.) of the 29 patients, and none from the others ; re-examinations of 26 of the patients at monthly intervals for 17 months revealed that 15 of them still harboured eggs in the rectal mucosa and the other 11 did not, so the cure rate as judged

by rectal biopsy was 42 per cent. In another group of 39 infected persons similarly treated, 30 of whom were also followed up for 17 months but in this case by stool examination as the test of freedom from infection, only 33 per cent. were found apparently to be cured ; the authors conclude that stool examination proved a more reliable means of assessing the presence of ova than did rectal biopsy in their hands. [See also GELFAND, this *Bulletin*, 1950, v. 47, 376.] *A. R. D. Adams*

WRIGHT, W. H. **Bilharziasis as a Public-Health Problem in the Pacific.** *Bull. World Health Organization.* Geneva. 1950, v. 2, No. 4, 581–95. [33 refs.]

Although [Eastern] bilharziasis is endemic in China and in the larger islands of the Western side of the Pacific its public health significance is inadequately appreciated and the diagnosis in individuals is often overlooked. This is largely because the condition does not appear in spectacular outbreaks, such as are associated with the epidemic diseases prevalent in these regions. Though there were only two deaths in 1,700 cases of [Eastern] bilharziasis among the American forces on Leyte island of the Philippines, it is estimated that 300,000 man-days were lost to the army through this disease and the local medical expenditure on it amounted to three million dollars. Among the indigenous populations of the endemic areas bilharziasis is allowed to pass into the chronic debilitating form which renders its victims a ready prey to intercurrent infections.

The author has already defined the known geographical distribution of *Schistosoma japonicum* and its vectors [this *Bulletin*, 1948, v. 45, 913] ; to this list he now adds a number of other areas of endemicity in the Philippines, and he gives further information on the incidence and distribution of the disease in China and Japan. An estimated 33 million persons throughout China suffer from Eastern schistosomiasis, and there are 174 thousand affected in Japan and some quarter of a million in the Philippines. The methods of computation of the number of those affected in these countries are indicated, and it is evident that that for China is a highly speculative one.

The nomenclature applied to the snails reported by various workers to be efficient vectors of the worm is confused ; it seems clear that all the known natural vectors belong to the genus *Oncomelania*, and that most if not all of the recorded vector species are synonymous with *O. hupensis* or *O. nosophora*. One cannot be too arbitrary and exclude the possibility that other species of *Oncomelania* can serve as vectors, particularly in view of the recent demonstration in the U.S.A. (BERRY & RUE, 1948, *J. Parasitology*, Suppl. v. 34, 15) that *Pomatiopsis lapidaria* can act in this capacity. The known vector snails are amphibious, spend most of their time in water, favour moist soil at the edges of slow-moving watercourses rich in vegetation, and are not found in the larger swiftly-flowing streams ; at the northern limits of their distribution they hibernate during the winter in crevices in soil above the water line. They are not usually found in the rice-fields proper, but may be located around their margins.

Details [which should be consulted in the original] are given of the ecology and development of the known snail vectors in the northern and the southern zones of endemicity, and these have a bearing on the times of the year when molluscicides most profitably can be applied. In the northern zones, snail destruction is most effective in the spring and the autumn, but in the southern zones it should be attempted after the rainy season and again towards the end of the dry season.

Far Eastern schistosomiasis is largely an occupational disease of agriculture ; its spread is favoured by lack of sanitation and by the use of human faeces

as fertilizer, particularly in rice cultivation. Swine, dogs and cats, oxen and buffalo, and rats all may harbour the infection, and so may play a part in its maintenance and dissemination. The methods of control advocated include :—

" 1. Chemical treatment or suitable storage of night-soil to render ova non-viable.
2. Paving of drainage ditches and irrigation canals and removal of vegetation from banks to facilitate the flow of water.
3. Large-scale treatment of infected individuals.
4. Control of infection in reservoir hosts.
5. Education of the public to limit the practice of promiscuous defaecation and the custom of bathing and washing clothes in infested waters.
6. Use of molluscicides."

The discovery of an effective, cheap and safe chemical means of destruction of snails remains still to be made. This would afford a means of attack at the weakest point in the cycle of propagation of the parasite. A start has been made with the successful trials of dinitro-*o*-cyclohexylphenol and its dicyclohexylamine salt [this *Bulletin*, 1947, v. 44, 919], which destroy not only the snails but also both eggs [? of the parasite] and cercariae. *A. R. D. Adams*

VAN GREMBERGEN, G. Le métabolisme respiratoire du Trématode *Fasciola hepatica* Linn., 1758. [**The Respiratory Metabolism of** *F. hepatica*.] *Enzymologia.* The Hague. 1949, Apr. 26, v. 13, No. 5, 241–57. [22 refs.]

SCHEID, G. & MENDHEIM, H., with R. AMENDA. Die Lanzettegelinfektion (Dicrocoeliasis) beim Menschen nebst Mitteilung eines neuen Falles. [**Dicrocoeliasis in Man with an Account of a New Case.**] *Ztschr. f. Tropenmed. u. Parasit.* Stuttgart. 1950, July, v. 2, No. 1, 142–50, 1 chart. [44 refs.]

The small liver fluke (*Dicrocoelium dendriticum*) has been found in man more rarely than has *Fasciola hepatica*, of which there are probably about 300 references in the literature.

Both parasites are found in ungulates as well as in rodents and in monkeys.

The clinical picture of dicrocoeliasis in man is not exactly clear. On the whole the liver appears to be enlarged, with secondary anaemia and high eosinophilia.

This present case was diagnosed in a Munich orphanage in an 8-year-old boy suffering from oxyuriasis.

The eggs of *D. dendriticum* were found in a cellophane scraping from the perineum. The liver was enlarged three finger breadths below the costal margin. No eggs were demonstrated in the duodenal secretion.

After administration of decholin eggs appeared in the faeces. Treatment consisted of 6 injections of Fouadin beginning with a dose of 1·5 cc. ; and 2·5 cc. was well tolerated in the next 3 days, but the last injections at this dosage tended to cause vomiting and the drug was stopped.

An intracutaneous test was devised with the use of the infected sheeps' livers as antigen, with controls. In titres of 1 : 100 ; 1 : 500 and 1 : 1000 a wheal developed and was thought to be diagnostic.

In this case injections of Fouadin were followed by a rapid tenfold increase of the egg count succeeded by a rapid decrease and then disappearance of the eggs from the faeces on the 10th day of treatment. *P. Manson-Bahr*

PARANDE, A. S. **A Case of Intestinal Infestation with** *Hymenolepis diminuta* **in Man.** *Indian Med. Gaz.* 1950, June, v. 85, No. 6, 256, 2 figs. on pl.

Prieto Lorenzo, A. Estudio parasitologico de la uncinariasis y su distribución geografica. [**Parasitological Study of Hookworm Disease and its Geographical Distribution.**] *Med. Colonial.* Madrid. 1950, Sept. 1, v. 16, No. 3, 190–218, 11 figs. & 1 map. [70 refs.]

A general study.

Boari, D. Alcuni rilievi e ricerche sullo stato attuale delle parassitosi intestinali con particolare riguardo all'anchilostomiasi nel territorio del comune di Cremona [**Research into the Distribution of Ankylostomiasis and of other Intestinal Parasites in the District of Cremona, Italy.**] *Ann. d. San. Pubblica.* Rome. 1950, Mar.–Apr., v. 11, No. 2, 459–97, 13 figs. & 1 plan. [25 refs.] English summary.

Ankylostomiasis was made notifiable in Italy in 1923, only in the case of certain occupations, such as miners, brickmakers, tunnellers and other underground workers. In 1933, compulsory notification was extended to all cases, actual or suspected. Boari, who is a health officer for Cremona and is also attached to the Institute of Hygiene of Parma University, has now carried out a survey of the district of Cremona, which includes a central urban area, some suburbs and an extensive surrounding rural area. For his investigations, he selected 2 brickworks, a small farmhouse, 2 town gardens and the refuse disposal works and depot which are some distance outside the urban area. He searched for helminth ova in the faeces and studied the blood of brickmakers (170), peasants and their families (95), market gardeners and their families (38), town gardeners and their families (27) and workers at the refuse depot (15)—345 persons in all. The personal habits of these persons, and their working or living environment, were also investigated, specimens of soil, clay, refuse heaps, etc., being examined for evidence of helminths. Personal habits were found to be primitive in many cases. At the brickworks, good latrine facilities are provided and maintained, but a large number of the labourers prefer to use the open ground. In the rural area generally, primitive latrines with reed shelters are often provided and misused. Ditches are made use of for latrine purposes when dry : the water supply is dependent on shallow wells : middens and privies are common. Eating habits are primitive and the hands are not washed before a meal. Children go about barefoot and play on the damp ground of the yards that are to be found in farmhouses. Labourers use only " open " clogs for footwear. Town refuse is pulverized and the heaps are then allowed to ferment for several months, after which the material is sold for use on the soil. Much organic manuring is done on the fields.

In spite of improved economic conditions, living habits are of a low standard, and nutrition included vegetables, eggs and dairy products with very little meat.

About half of those infested with helminths show no sign of illness. In the remainder, complaints are slight ; only about 1·7 per cent., can be considered ill as a result of harbouring the helminths. The presence of the infestation has been often missed even when anaemia is present in the inhabitants of this district.

The problem calls for a sound programme of health education and environmental improvement, together with a thorough search for cases and their treatment. Boari considers that the infestation may lead to under-development and infantilism if it is present in childhood or adolescence.

[Those specially interested in this subject will like to read the full article for its statistical and other details.] *J. Cauchi*

SOLETTA, L. Osservazioni e ricerche sulla presenza della anchilostomiasi nella provincia di Parma. [**An Ankylostomiasis Survey in the Province of Parma, Italy.**] *Ann. d. San. Pubblica.* Rome. 1950, Mar.–Apr., v. 11, No. 2, 499–513, 2 figs. [12 refs.] English summary.

Following the recent notification of two cases of this helminthic infestation in market gardeners from Valera, which is just outside the city of Parma, Soletta has carried out a pilot survey in a few selected localities.

From market gardens in Valera, a total of 5 households and 103 persons are reported upon ; 3 of these homes are clean and the occupiers are in the habit of washing their hands at the end of their [market gardening] work ; 4 latrines of poor construction serve 14 families ; the yard is of beaten earth and well kept. In the vegetable garden, close to the house, there is a manure heap standing on pervious soil. Water from a ditch is used for the washing of vegetables in an iron tank. Gardening is done in bare feet. A fourth house is modern in construction, with good ventilation and lighting, and has a good pipe-borne water supply for the washing of vegetables. The remaining household which was examined is in an unsatisfactory building and presents a low standard of nutrition, cleanliness and education; the latrine and the manure heaps are insanitary : children play in the vegetable garden because there is no yard. Twenty-five persons, representing 17·7 per cent. of the men and 29·3 per cent. of the women, were found infested. There were hardly any cases among children or old people. Cesspool contents from both town and outside brickworks are widely used as manure.

Also in Valera, 102 employees of a paste [macaroni] factory were examined and two women and one man were found infested ; these 3 persons had recently been employed in market gardening. Factory equipment, processes and sanitary facilities were all up-to-date.

For comparison, a market garden has been investigated at Cortile S. Martino in another part of the province of Parma. Here, cesspool contents from a nearby " Parmensi " brickworks are used as manure. The home conditions are good, the latrine well kept and manure is kept on a concrete platform. Water supply is from a deep tube well and this water is used for laundry and vegetable washing purposes. The people show a good standard of education, but wear no shoes or other covering for their feet during gardening. No case of hookworm infestation was found in this family.

The nearby " Parmensi " brickworks, though lacking the most modern equipment, is well run on hygienic lines and machines are used for digging the clay, transporting it and making it into bricks. The workmen are provided with footwear and their standards of nutrition and education are good. They use the latrines provided, but these are badly kept. The claypit is very deep, reaching ground water, and the workmen driving the excavators are provided with gum boots. The only case of hookworm was found in the foreman who had contracted it elsewhere.

Examination of the blood from persons who were found infested showed eosinophilia of a high degree in 11, moderate in 17 ; hypochromic anaemia in 13, normochromic in 15 with no hyperchromic cases : the red blood count was 3·5–4 million per cmm. in 16, 4–4·5 million per cmm. in 8, and 4·5–5 million per cmm. in 4.

A search for evidence of helminths in 10 specimens of soil obtained from the market gardens and from the brickworks proved negative, as did the examination of soil washed off the vegetables ; this may have been due to the late season [dates not stated] when these investigations took place.

Enquiries into the literature and into local hospital records revealed that 49 cases of ankylostomiasis were diagnosed between 1889 and 1909 and 5 more

cases during 1909–1942. It is possible, however, that many other cases recorded as anaemia without other specification may have been due to hookworm infection.

Cesspool contents should be rendered harmless by adequate treatment, *e.g.* 20 per cent. ferrous sulphate, or their use as manure discouraged by cheapening the sale of fertilizers of the chemical type, such as cyanamide which has the further advantage of having disinfesting powers. The custom of working in bare feet needs reforming. *J. Cauchi*

LINDQUIST, W. D. **Some Abnormal Host Relationships of a Rat Nematode, *Nippostrongylus muris*.** *Amer. J. Hyg.* 1950, July, v. 52, No. 1, 22–41, 21 figs. on 4 pls. [16 refs.]

VAN GREMBERGEN, G., VAN DAMME, R. & VERCRUYSSE, R. Le métabolisme respiratoire du nématode *Ascaris lumbricoides*. [**The Respiratory Metabolism of *A. lumbricoides*.**] *Enzymologia*. The Hague. 1949, Dec. 27, v. 13, No. 6, 325–42, 4 figs. [30 refs.]

ARONSTAM, E. M. **Acute Abdominal Manifestations of Ancylostomiasis. Report of Two Cases.** *U. S. Armed Forces Med. J.* 1950, Aug., v. 1, No. 8, 935–7.

POINDEXTER, H. A. **Filariasis Bancrofti Studies in Liberia.** *Amer. J. Trop. Med.* 1950, July, v. 30, No. 4, 519–23, 1 graph.

Malaria and *Wuchereria bancrofti* infections are found in every part of equatorial West Africa, but the incidence of either varies greatly in different parts, including Liberia. During the last two and a half years survey teams of the U.S. Public Health Service Mission have made sample studies in three provinces and four out of the five counties of Liberia. In the determination of the distribution of these two groups of diseases thick blood smears have been taken and studied from over 46,000 Liberians during that period. It is estimated that there are between 25,000 to 30,000 persons with microfilariae in their blood in Liberia, and this, with several reservations, constitutes a reliable index of the clinical prevalence of the disease. The table which has been constructed demonstrates that night smears are 13 times more efficient than day smears for laboratory diagnosis and that the disease is more prevalent in the southern sections of the country. Evidence was obtained of the importance of repeated exposures to the bites of infected mosquitoes in the development of clinical *W. bancrofti* infections by the fact that not a single positive smear for microfilariae was found in 100 Government officials or their families in one town who lived apart from native quarters and employed some anti-mosquito measures, even though 32 per cent. showed *Plasmodium falciparum*. In contrast to this, 850 Liberians in the same town showed 16·7 per cent. positive blood smears for microfilariae. Though night smears are so much more effective than day smears in the laboratory diagnosis of microfilaraemia, young adults with heavy infections showed a few microfilariae in blood smears taken during the day especially in the early mornings.

During the last two and a half years hourly microfilaria counts have been made during one-day periods on 10 young adult males between the ages of 18 and 30. One curve was obtained from one Liberian of 18 years and this exhibited an anadicrotic wave, or a notch occurring in the count between 4 p.m. and 6 p.m. daily : 16 drops (or 1 cc.) of blood were used for each determination. As the anadicrotic wave persisted, a second series of 9 men were studied ; this anadicrotic wave was exhibited at some time in the afternoon or early evening in this case also.

An occasional case of elephantiasis as a complication of *W. bancrofti* infection may be over-emphasized, and lead to disproportionate expenditure of public funds on filariasis control. *P. Manson-Bahr*

DEANE, Maria P. Sôbre a incidência de filárias humanas em Manáus, Estado do Amazonas. **Human Filariasis in Manáus, State of Amazonas, Brazil.** *Rev. Serviço Especial Saúde Pública.* Rio de Janeiro. 1949, July, v. 2, No. 3, 849–58, 1 map. English summary.

This investigation was carried out between June and September 1948 ; the town was divided into 18 zones as in a previous inquiry into malaria. Altogether 2,405 persons were examined, about 3·5 per cent. of the population ; the examination was by thick drop taken between the hours of 7 and 9.30 p.m. From any found positive for filarial embryos more slides were made for studying the morphology and determining the species of the microfilaria. In 48 (2 per cent.) of the smears embryos of *W. bancrofti* were seen and in 15 (0·6 per cent.) those of *Mansonella ozzardi* ; no instances of dual infection were found. In a table are given the numbers examined in each zone, the numbers and percentages positive for either parasite and the number probably autochthonous. Two other tables give the incidence by age of the whole population and of the autochthonous population.

Culicine mosquitoes, 936 altogether, were captured in 176 houses. These were not dissected for the developing embryos, but 89 per cent. of them were *Culex fatigans*, the remaining 11 per cent. including *Anopheles darlingi*, *A. albitarsis*, *A. pessoai*, *A. nimbus*, *Culex ocellatus*, *Psorophora albipes*, *Taeniorhynchus fasciolatus* and *T. arribalzagae*. The numbers and percentages of these are shown in another table, but in the cases of four of them a single specimen only was caught, in another only two, so the figures convey little. Most of the positive autochthonous infections by the filaria were in the São Raimondo district of the town. The infection rate was about the same in all districts for the different age-groups and the inference is drawn therefrom that the infection is of recent introduction. The majority, 8 out of the 15, of the *M. ozzardi* infections appeared to have been contracted outside Manáus.

H. Harold Scott

DEANE, Maria P. Sôbre o valor do exame de sangue em gota espêssa colhida às primeiras horas da noite para inquéritos de filariose de Bancroft. **The Value of Examination of Thick Smears of Blood taken in the Early Hours of the Night in determining the Incidence of Filariasis.** *Rev. Serviço Especial Saúde Pública.* Rio de Janeiro. 1949, July, v. 2, No. 3, 859–63. English summary.

The author, when investigating the incidence of filarial infection in Manáus, Brazil, took thick drop specimens of blood in the early hours of the night, 7 to 9.30 p.m. [see preceding abstract]. As this was considerably earlier than was usual for looking for filarial embryos it was possible that a number of cases might be missed. To test this, the author examined the blood of 968 persons in Belém, Pará State, blood being taken between 7 and 9.30 p.m. as before, and compared the results with those found in larger quantities of blood (2 cc.) by Knott's method and taken later, between 11.30 p.m. and 3 a.m. Of the total 968 there were 121 (12·3 per cent.) positive in the early hours' specimens and 139 (14·3 per cent.) by the Knott method—a difference of only 2 per cent., not enough to be significant, nor to warrant the inconvenience of disturbing the patient in the middle hours of the night. *H. Harold Scott*

PÉREZ ARA, A. Filariasis del epididimo. Reporte de una primera observación en Cuba. **Filaria in the Epididymis. First Case recorded in Cuba.** *Rev. Kuba Méd. Trop. y Parasit.* 1950, Mar.–Apr., v. 6, Nos. 3 4, 40–45, 9 figs. [12 refs.] English summary.

A man of 21 years had suffered from pain and swelling of the left testicle at intervals during the preceding 8 years. A month ago he was treated with penicillin for an attack of gonorrhoea. The specimen sent for histological examination had been removed at an operation for epididymectomy. On making sections of the swollen head of the epididymis the authors found two, perhaps more, filariae. Embryos were not seen in the patient's blood—the worms may have been dead for some time—so the species was not determined with certainty, but was believed to be *W. bancrofti*. The pathological histology is described in detail and depicted in a series of photomicrographs.

Filariasis seems to be rare in Cuba : three instances of its having been found at operation are noted : in 1915 at an operation for right inguinal hernia ; in 1927 in a small tumour of the spermatic cord, and in 1946 in a "lymphatic cyst of the left cord." The present is the first in which it has been found in the epididymis. *H. Harold Scott*

WILSON, T. **Hetrazan in the Treatment of Filariasis due to *Wuchereria malayi*.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1950, July, v. 44, No. 1, 49–66, 6 charts & 1 diagram. [12 refs.]

The investigations took place at Sungei Patani in the north-west corner of the Malay peninsula where an endemic area of filariasis "*W. malayi*" occurs. Doses of Hetrazan were recorded as the citrate salt, which contains 51 per cent. base. The patients were mostly symptomless carriers of microfilariae who were persuaded with difficulty to enter hospital for treatment, and who accordingly would take a serious view of any untoward reactions produced by the treatment. The first three patients had microfilariae in the blood but no symptoms. They received 1·7–2·15 mgm. per kgm. thrice daily for 21 days. They developed a severe reaction within 6 to 12 hours of the first dose—pyrexia (103°F.) severe headache, nausea and vomiting. The symptoms abated considerably by the 3rd day and disappeared completely by the 5th. A fourth patient with symptoms of filariasis but no microfilariae, who received the lowest dose (1·7 mgm. /kgm. thrice daily), had no reaction except a mild urticarial rash on the 6th and 7th days. The microfilarial count diminished dramatically ; two patients whose night blood contained 1,200 and 750 microfilariae per 60 cmm. had only 6 microfilariae after receiving three doses in 9 hours. In the second group of three patients, the dose was 1·1–1·6 mgm. per kgm. thrice daily. The febrile reactions were similar but a little less intense and prolonged ; the effect on the microfilariae was the same.

Including the cases described above, Hetrazan was given in all to 20 patients who had microfilariae but no symptoms of filariasis. The initial doses ranged from 0·28 to 2·15 mgm. per kgm. thrice daily. All had fairly severe reactions as described above, although the reactions were a little milder if the dose was below 1·0 mgm. /kgm. Once the reaction had subsided, the dose could be quadrupled without ill-effect. The microfilaria count fell rapidly and in 15 out of 16 patients it became zero at the end of treatment. In two patients, 0·12 mgm. per kgm. thrice daily was given ; this dose produced no reaction but had no effect on the microfilariae. When the dose was raised to 0·25 mgm. /kgm. the microfilaria count fell slowly and a mild reaction took place.

Eight patients with clinical filariasis but no microfilariae were treated. Initial doses of 1·5 to 2·3 mgm. per kgm. were given without provoking any

febrile reaction, except for one boy who on the 5th day of treatment (when on a dosage of 3·6 mgm./kgm. t.i.d.) had an attack of filarial fever with pain and swelling of the right groin and leg. There was no significant improvement in the symptoms of filariasis. One man was treated who had both clinical symptoms and microfilariae ; he was given a total dosage 108 mgm. per kgm in 15 days. The microfilariae disappeared from his blood and the swelling of his leg disappeared. Half of the microfilaria carriers passed round worms during treatment. During the febrile reaction the number of neutrophile leucocytes in the blood temporarily rose, while that of eosinophiles temporarily fell. About 12 months later, 6 out of 11 carriers of microfilariae were still free from microfilariae while the others showed only much reduced counts. Four of the patients with clinical symptoms were re-examined 10 months later. All reported improvement in physical conditions and in ability to work uninterrupted by filarial fever. The reactions produced in these patients were much more severe than those described by other authors as occurring in patients with *W. bancrofti* : the possibility is discussed, whether this greater severity is due to the different species of worm involved. *F. Hawking*

Gordon, R. M., Kershaw, W. E., Crewe, W. & Oldroyd, H. **The Problem of Loiasis in West Africa with special reference to Recent Investigations at Kumba in the British Cameroons and at Sapele in Southern Nigeria.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1950, July, v. 44, No. 1, 11–41, 2 maps & 2 pls. [74 refs.] Discussion 41–7 [Shortt, H. E. (President) ; Buxton, P. A. ; Hawking, F. ; Chesterman, C. C. ; Kirwan, E. O'G. ; McRobert, G. ; Macdonald, G. ; Gordon, R. M. & Kershaw, W. E. (in reply)].

This is a general account of the problem of loiasis with particular reference to the work done by Gordon and his colleagues at Kumba in the British Cameroons where a research station was established in 1949.

As far as is known at present, full development of microfilariae of *Loa loa* occurs only in *Chrysops silacea*, *C. dimidiata*, and *C. distinctipennis*. Apart from two doubtful exceptions, the disease is apparently confined to the equatorial rain forest of Africa which extends from 8°N. to 5°S. of the equator, and from the Gulf of Guinea eastwards to the Great Lakes. The genus *Chrysops* is represented in almost all parts of the world but the subgenus *Kleineana*, which contains all the known and suspected vectors of *Loa*, is limited to Africa. *C. silacea* and *C. dimidiata* are restricted to the rain forest region and in this part there is little doubt that they are the only important vectors ; at the fringes of the forest it is possible that other species, *e.g.*, *C. distinctipennis* in the southern Sudan, may act as vectors. *C. longicornis* probably does not transmit.

At Kumba, among 2,000 adult flies collected, all were *C. silacea* or *C. dimidiata*, but when larvae were gathered and reared they produced also *C. longicornis*, *C. langi* and *C. griseicollis*, none of which had been known to occur at Kumba. This suggested that they must live disassociated from man, feeding presumably on animals, among which monkeys might form a reservoir of filarial infection. The larvae of *Chrysops* are found in densely shaded streams where the course is impeded by vegetation, the flow is slow and the bottom is covered by decaying leaves over soft mud, overlaid by fine sand. The adult fly bites chiefly between 8.30 a.m. and 5.0 p.m. but it is averse to feeding in full sunlight. The adult females of *C. silacea* and *C. dimidiata* normally live in the forest canopy. Europeans at Kumba are bitten mostly in the shade of their houses, which

stand on a high ridge at the level of the tops of the high trees growing in the adjacent valleys, so that they form interruptions in the forest canopy. *Chrysops* seldom enter the houses of Africans, which are lower in position. The female flies entering the houses probably hunt by sight and are attracted by moving objects. In the forest round Kumba, visibility at ground level is severely restricted by vegetation ; therefore *Chrysops* is not attracted down from the forest canopy and the impression is given that *Chrysops* is absent from such areas. When a platform was erected 50 ft. up a tree, or when a clearing was made in the undergrowth below it, specimens of *C. silacea* were readily captured by fly boys. On the rubber plantations at Sapele in S. Nigeria the picture is quite different. Both species of *Chrysops* feed voraciously throughout the plantation area and enter well-lighted European houses (while avoiding dark African houses). This is because the rubber trees are low-growing and persons on the ground can easily be seen by flies in the canopy above. Male *Chrysops* do not feed on blood, they have not been captured during these studies, and their habitats are still unknown.

When *Chrysops* bites, it does not introduce any toxin into the skin ; any reaction which may occur is possibly due to the person having become sensitized by previous exposure either to the insect's saliva or to the larvae of *Loa*. Among 22 of 65 adult Africans examined at noon the density of microfilariae varied between 1 and 1,500 per 50 cmm. of blood. *Chrysops* feeds from a pool of blood which collects when the tissue is lacerated by the proboscis. The average amount of blood taken by *C. silacea* in one meal is about 10-40 cmm. The average infection rate among wild flies at Kumba is about 7·2 per cent. The flies probably feed about once a fortnight, and as development of the worm takes about 12 days, they usually have only one infection (one brood) at a time. Although many worms are often present in an infected fly, they do not seem to have an adverse effect on it. When an infected fly feeds, large numbers of infective larvae emerge and are deposited on the surface of the skin, from which they disappear rapidly. The exact way in which the larvae penetrate the skin is, however, still obscure. The development of the adult worm in man probably takes between 6 and 18 months but practically nothing is known of this phase of the life history. The fully adult worms may occur in almost all anatomical regions, but are always limited to connective tissue. In man we do not know whether their rapid translatory movements occur elsewhere than in the subcutaneous tissue and conjunctiva but in monkeys similar worms have been seen moving along intermuscular septa and similar fascial planes. Many patients with loiasis appear to have no microfilariae in their blood. In a large proportion of these cases, microfilariae are really present in small inconstant numbers and can be detected by repeated examinations. In the remainder, Gordon suggests that the microfilariae may accumulate in a reservoir in the internal organs.

The extent of the reaction in the human host varies greatly in different individuals. Calabar swellings appear to be allergic reactions due to products of the living or dead worm. Possibly repeated superinfection leads to some degree of immunity to further invasion by worms. Treatment of loiasis is best done with Hetrazan. The effective control of loiasis requires the accumulation of much more information. D.M.P. [dimethyl phthalate] (60 per cent.) is a satisfactory repellent to *Chrysops*. Extensive clearing of the forest would probably be effective but would be expensive. It is doubtful whether spraying with DDT would be effective in destroying the adult flies. Methods for control of the larvae will probably be feasible but they still require more investigation. Various points raised in the paper were amplified in the subsequent discussion. [This is an interesting and detailed paper which contains more information than can be conveyed in an abstract.] *F. Hawking*

Meneses Rubiánez, C. & Avendaño Escalante, S. Estudio comparativo entre los metodos de Hall y Graham para el diagnostico de la oxyuriasis. [**Comparison between the Methods of Hall and of Graham in the Diagnosis of Enterobiasis.**] *Rev. Chilena Hig. y Med. Preventiva.* 1949, Mar., v. 11, No. 1, 45–7.

The authors have compared the results obtained by Hall's Cellophane strip and Graham's transparent Scotch cellulose tape methods [this *Bulletin*, 1941, v. 38, 525] for the diagnosis of infestation by *Enterobius vermicularis*. They tried both methods on 100 children of the Municipal Homes for Boys and Girls at Viña del Mar, Chile. Each method was used for each child at an interval of 8 days. Forty-eight were positive by Hall's method, 88 by Graham's and in 36 of the 52 negative to Hall's the results by Graham's method were positive. Of 45 girls 33 were positive ; of 55 boys 45 were positive, a difference not statistically significant. The authors conclude : The sticky cellulose tape is more adhesive than the Cellophane strip for the ova of *Enterobius vermicularis*, thus increasing the likelihood of a positive finding ; the field for examination is smaller and easier to examine, owing to the folds in the Cellophane ; the ova adhere and are not destroyed, and, finally, the preparation can be kept indefinitely and repeatedly examined, if necessary. *H. Harold Scott*

Anselmo, J., Bahamondes, S., Ipinza, I., Lagos, R. & Cortés, G. Contribución al estudio de la triquinosis inaparente. [**Studies of "Inapparent" Trichinosis.**] *Rev. Chilena Hig. y Med. Preventiva.* 1949, Mar., v. 11, No. 1, 51–3, 1 fig.

The English summary appended to the paper is as follows :—

" Looking for cysts of *Trichinella spiralis*, the authors have examined 135 corpses in two hospitals of Santiago. They found the parasites in 5 (3·7%)."

DEFICIENCY DISEASES

Cullumbine, H., Bibile, S. W. & Wikramanayake, T. **A Study of the Diets of various Ceylonese Communities.** *Ceylon J. Med. Sci.* (Sect. D.) 1949, Dec., v. 6, Pt. 4, 255–86. [20 refs.]

Janssen, E. & Le Roux, J. S. **The Syndrome of Malignant Malnutrition. Observations on the relation of the Serum Proteins to the Occurrence of Oedema and the Effect of Diet.** *South African J. Clin. Sci.* Cape Town (incorporating *Clin. Proc.*). 1950, June, v. 1, No. 2, 100–113. [54 refs.]

This paper is based on a therapeutic study of 101 cases. A first group of 50 children received the normal hospital diet, including skimmed milk, with desiccated hog's stomach. The second group received skimmed milk with calcium caseinate. Both groups were given numerous vitamin supplements and many received liver, either fresh or as extract by injection.

On admission all patients had oedema with reduced serum protein levels, the average total protein being slightly over 4 gm. per 100 ml. with an albumin / globulin ratio slightly under unity. Death occurred in 18 of Group I and in 13 of Group II. Infections were common and were considered to be the cause of death in six cases in each group.

With treatment serum protein levels returned to normal, more rapidly in Group II, and oedema diminished and finally disappeared, but no consistent relationship was found between serum protein levels and the severity or rate

of disappearance of the oedema. On these grounds it is considered that hypoproteinaemia, due to a low dietary protein intake, though a fundamental feature of the syndrome, is not the only cause of the oedema.

Very serious cases, and those with severe anorexia, amounting often to complete refusal of food, were treated successfully by continuous gastric drip. It is recorded that intravenous administration of blood, serum, saline or protein hydrolysates proved useless and dangerous [*cf.* PIERAERTS, this *Bulletin*, 1950, v. 47 265, who found parenteral administration of human plasma a life-saving measure in apparently similar cases.] *Dean A. Smith*

BLACK, R. H. **Hepatomegaly in the Natives of Northern Australia.** *Med. J. Australia.* 1950, July 1, v. 2, No. 1, 10–12, 1 fig. (map).

In the course of a malaria survey, 1,382 natives of the northern part of Western Australia were examined and liver enlargement was found in 249 (16·5 per cent.). The livers were firm and regular: no cases of jaundice or ascites were seen. Hepatomegaly commonly occurred at an early age in these people (14 per cent. of children under 10 years), and persisted throughout life, a slight increase in size occurring with increase in age.

In considering the probable aetiology of the condition the author notes that only 0·6 per cent. of the population had palpable spleens, and no malaria parasites or microfilariae were found. Kala azar, schistosomiasis and hydatid disease have not been reported in the area. Ova of hookworm (3 cases) and of *Hymenolepis nana* (2 cases) were found in 23 stool specimens examined.

It seems likely that the hepatomegaly is due to chronic subnutrition though further investigations are necessary before the aetiology can be established.

Dean A. Smith

STRANSKY, E., DAUIS-LAWAS, Domiciana F. & VICENTE, C. **On Ascorbic-Acid (Vitamin-C) Deficiency in the Tropics.** *J. Trop. Med. & Hyg.* 1950, Aug., v. 53, No. 8, 170–74. [37 refs.]

Ascorbic acid estimations were performed upon specimens from three groups of persons in the Philippines. (1) The average value for 20 samples of breast-milk was 3·7 mgm. per cent., ranging from 1·0 to 9·5 mgm. per cent. (2) The mean value for plasma-ascorbic acid level of 89 normal nursing mothers was between 0·5 and 0·55 mgm. per cent., ranging from under 0·1 to over 1·4 mgm. per cent. (3) Of 124 children seen in the hospital paediatric department, 15 had plasma levels below 0·1 mgm. per cent., 79 had plasma levels below 0·5 mgm. per cent., and 111 below 1·00 mgm. per cent. Only one case of clinical scurvy has been seen in 11 years but the authors consider that chronic ascorbic acid deficiency is an important cause of lowered resistance to infection.

Dean A Smith

HUTTON, P. W. **A Case of Pellagrous Neuropathy.** *East African Med. J.* 1950, Aug., v. 27, No. 8, 325–7.

"A case of severe neuropathy of the spinal cord is described in association with a severe nicotinic acid deficiency and minor pellagrous skin changes.

"The nature of the case suggests rather that the deficiency was induced by some unknown factor than by dietary or digestive inadequacy."

HAEMATOLOGY

LIPPI, M. & ARGIOLAS, N. Ricerche ematologiche in adulti sardi non malati. [**Haematological Studies of Healthy Adult Sardinians.**] *Arch. Ital. Sci. Med. Trop. e Parassit.* 1950, Mar., v. 31, No. 3, 202–17. [61 refs.]

The English summary appended to the paper is as follows :—

" From tests on the blood crasis of Sardinians, certain points appeared to disagree with known and recognised facts. Hypochromy was more or less observed in all cases, together with a tendency to microcytosis, a remarkable percentage of oval erythrocytes, poikilocytes, and " target " element.

Alterations in the resistance were further noted, showing an increased distance between the higher and lower values ; often the bilirubin content of the blood in its indirect reaction appeared also increased. The number of leukocytes maintained itself usually at the physiological minimum, with relative lymphocytosis and frequent deviation to the right of Arneth's scheme.

The alteration of the red blood elements agree with the opinion of the authors according to which a haemolytic constitution seems to prevail in the Sardinians, which constitution might possibly be due to influence of malaria."

HODGES, J. H. **The Effect of Racial Mixtures upon Erythrocytic Sickling.** *Blood.* 1950, Sept., v. 5, No. 9, 804–10, 3 figs. [15 refs.]

" The incidence of erythrocytic sickling is less in " pure " Negroes than in those with small admixtures of white and American Indian ancestry.

" There is no evidence that the dilution of Negro ancestry, to such an extent that the person is one-half or less Negro, will either increase or decrease the incidence of sickling."

DEMARSH, Q. B., with the technical assistance of Susan BOYCE. **Severe Mediterranean Anemia (Cooley's Anemia) in a Chinese Child.** *Blood.* 1950, Sept., v. 5, No. 9, 798–803, 5 figs. [11 refs.]

" 1. The fifth known case of Cooley's anemia in a Chinese child is reported. Death occurred following a febrile illness which was thought to be mumps encephalitis. Autopsy findings were consistent with this diagnosis and with Cooley's anemia.

" 2. The remainder of the family showed the characteristic blood findings of Cooley's trait."

VENOMS AND ANTIVENENES

SWARTZWELDER, J. C. **Snake-Bite Accidents in Louisiana : with Data on 306 Cases.** *Amer. J. Trop. Med.* 1950, July, v. 30, No. 4, 575–89, 1 fig.

Of the many species of snakes in Louisiana, more than 40 in number, the following six are poisonous to man : the copperhead (*Ancistrodon makasen*) which is more active at night ; the cottonmouth moccasin (*Ancistrodon piscivorus*) ; the pigmy rattlesnake (*Sistrurus miliarus streckeri*) ; the canebreak rattlesnake (*Crotalus horridus atricaudatus*) ; the diamond-back rattlesnake (*Crotalus adamanteus*) and the coral snake (*Micrurus fulvius fulvius*), but there is no recorded case of a bite by this last.

The author analyses the records of 306 cases of snakebite during the forty years ending 1946. Only two proved fatal, one in 1914 and one in 1921 ; there had been no fatal cases during the past 25 years. More than four-fifths of the

cases occurred in the summer months, April to October. The ages of the patients showed a rise in the number of cases to the age of 15 and thereafter a fall, almost steady except for one quinquennium, 51–55 years, the successive quinquennial figures being 17, 44, 48, 40, 38, 26, 12, 18, 19, 7, 16, 6, 2, 5, 2 and one. The ages of 5 of the patients were not known. Twice as many whites as coloured persons were bitten. One of the fatal cases was that of a man who "professed to be a snake-charmer [who] was bitten by a rattlesnake whose venom glands he claimed to have removed previously. His ability to charm the snake apparently was equivalent to his surgical prowess". Rubber boots and overalls are not completely protective, since the fangs may penetrate them. Amateur first-aid treatment was at times drastic and had worse consequences than the untreated bite. A list of 114 fatal cases of "poisoning by venomous animals in Louisiana" between 1916 and 1945 is given, but beyond the vague statement that some were due to the Black Widow spider (*Latrodectus mactans*) the causes are not detailed. There is also a line map showing the distribution of deaths by parishes. Laboratory findings are mentioned, but were almost entirely negative. One patient had red corpuscles in his urine, but none had haemoglobinuria, nor any significant albuminuria; not even moderate anaemia or haemolysis; one-third showed leucocytosis, but this could be accounted for, in part at least, by infection. Treatment could not be evaluated because of the low general fatality rate and the main aim, at least one of them, should be to anticipate bacterial contamination of the wound. *H. Harold Scott*

Erratum

In this *Bulletin*, 1938, v. 35, 519, paragraph 4 of the abstract of the paper by Schöttler on the venoms of *Vipera latasti* and *V. lebetina* should read :—

"4. The active principle in each case is a powerful haemorrhagin. The anticoagulant action is much reduced by heating to 75°C. for 30 minutes and destroyed altogether at 100°C. for 5 minutes."

The comment by the abstracter, that the time intervals had not been given, should be deleted.

In this *Bulletin*, 1943, v. 40, 626, in the abstract of the paper by Schöttler, the first sentence of the second paragraph should read :—

"Mice were used as experimental animals; the toxin and antivenene were mixed and injected at once." [Not kept at 37°C. for ½ hour as stated in the abstract.]

The comment in the succeeding sentence should be deleted.

DERMATOLOGY AND FUNGUS DISEASES

Rodhain, J. **Pinta : (with reference to the Article by** R. D. G. Ph. Simons **entitled "Is Tinea albigena Nieuwenhuis a Separate Disease or an Indonesian Pinta combined with Epidermophytosis ?"). With remarks by** R. D. G. Ph. Simons. *Documenta Neerlandica et Indonesica de Morbis Tropicis*. Amsterdam. 1950, June, v. 2, No. 2, 166–9, 6 figs.

The article by Simons to which Rodhain refers was abstracted in this *Bulletin* [1949, v. 46, 1078]. Professor Rodhain thinks that the term "African pinta" is not altogether a happy one and may give rise to confusion because, so far as we know at present, pinta does not exist in Africa, but conditions of cutaneous depigmentation are fairly frequently encountered and in some cases at least the diagnosis is uncertain, but "pintoid yaws" is far from uncommon. The paper contains an excellent illustration of the latter in large patches and also

one with a multitude of small spots which started years before in small pruriginous lesions ; no mycelia or spirochaetes could be found and the diagnosis remains undecided. Simons in a comment agrees that yaws may cause non-atrophic leucoderma like pinta—hence his use of the term " pintoid yaws ". He reproduces more photographs showing affection of the hands and feet and mutilating lesions. The difficulties in separating the two diseases are that both are spirochaetal and in both the Wassermann reaction may be positive. " Positive serology does not confirm the diagnosis of pinta, but negative serology almost certainly excludes the diagnosis." *H. Harold Scott*

VARELA, G. & OLARTE, J. Anticuerpos contra el Treponema carateum del " Mal del Pinto " en el suero sanguineo de pacientes de esta enfermedad. [**Antibodies against** *Treponema carateum* **in the Serum of Patients suffering from Pinta.**] *Medicina.* Mexico. 1950, July 25, v. 30, No. 608, 316–17.

There is no cross-immunity between *mal del pinto* and syphilis ; syphilitic chancres may be seen in pinta subjects and *mal del pinto* can be transmitted to syphilitics. Attempts to inoculate pinta subjects with yaws are only partially successful. The authors have studied the effects of normal serum, syphilitic serum, and *mal del pinto* serum on the exudation of the skin of pinta patients rich in *T. carateum* and noted the results on the motility of the treponemata after 1, 2, and 3 hours. These are given in the following table :—

		Normal serum per cent.	Syphilitic serum per cent.	Pinta serum per cent.
First hour	Motile	100	90	75
	Non-motile ...	0	10	25
Second hour	Motile	100	65	35
	Non-motile ...	0	35	65
Third hour	Motile	70	20	15
	Non-motile ...	—	80	85

The authors conclude that in *mal del pinto* the antibodies immobilizing *T. carateum* " differ from the reagins which set up general serological reactions and that these antibodies against *T. carateum* are present in the serum of syphilitics." *H. Harold Scott*

LYON, E. **Remarks on Eosinophilic Erythroedema (Erythema Multiforme Perstans).** *Acta Med. Orientalia.* 1950, Jan., v. 9, No. 1, 17–23. [16 refs.]

Eosinophilic erythroedema is a clinical condition that has made its appearance since 1944 ; a few cases that fit this syndrome were, however, observed prior to this date. Most of the cases observed have been in middle-aged Jewish immigrants from Central Europe into Israel. The main clinical feature is transitory and migrating oedematous swellings involving the skin, or more rarely the mucous membranes, and the subcutaneous tissues. The skin is usually warm to the touch and has a reddish tinge ; there may also be " stripes or tortuous bands of redness ". There is at first pain and local tension, and pruritus which may persist for a considerable time. The swellings appear on various parts of the body and may " migrate " overnight to approximate or

distant parts. The swellings are often preceded by some generalized symptoms, neuralgia and malaise. The swellings may be temporary or they may persist for weeks or months, and they are subject to relapses after a year or more. There is usually an associated eosinophilia with or without a leucocytosis : this may not be present at the beginning but appears later.

A large and diverse variety of associated conditions have been reported by various writers : these include transitory pulmonary infiltrations, dry pleurisy and pleural effusion, periphlebitis, peripheral periarteritis, gall-bladder inflammation, temporary liver damage, epididymitis and a sensation of burning during micturition.

It has been suggested that drug sensitivity is an aetiological factor in this condition : three cases are quoted which appear to support this suggestion, but other cases have been observed in which the patient took no drugs. Amoebiasis and helminthiasis have also been considered as possible causes. The condition is regarded as allergic.

Temporary relief only has been achieved by the administration of antihistamine drugs.

It seems possible that permanent vascular damage occurs in certain cases.

L. E. Napier

Ghosh, L. M., Dey, N. C. & Panja, D. **Madura Foot (Mycetoma).** *Indian Med. Gaz.* 1950, July, v. 85, No. 7, 288–91, 4 figs. on pl. [30 refs.]

An account of 2 cases in Bengal.

TROPICAL OPHTHALMOLOGY

Gonnet, C. & d'Haussy, R. Notes sur l'ophtalmologie à Madagascar. Region des Hauts-Plateaux. [**Ophthalmological Conditions in the Hauts-Plateaux Region of Madagascar.**] *Méd. Trop.* Marseilles. 1950, Mar.–Apr., v. 10, No. 2, 253–72. [55 refs.]

These notes on ophthalmic diseases in the hilly districts of Madagascar are based on the authors' experience there during the four years.

They are of opinion that outbreaks of conjunctivitis are rare on the whole, very rare in the hilly parts although more frequent on the West Coast where it is warmer and dust and winds prevail.

Trachoma, formerly unknown in the island, has of recent years been imported and is found mostly on the West Coast in Indians and Chinese. The first autochthonous case was diagnosed in 1936. In marked contrast to the rarity of conjunctivitis, affections of the cornea are very common and serious. They comprise interstitial keratitis, hypopyon ulcer, rice beaters' keratitis and superficial keratitis of all types. The keratitis due to trauma by grains of paddy is seasonal and is responsible for a large amount of defective vision and blindness and could be prevented if more modern methods were employed in the threshing of the paddy. Relapsing fever occurs on a large area of the West Coast and, as in the case of other countries, the commonest ocular complication is irido-cyclitis caused by spirochaetes circulating in the blood stream : affections of the optic nerve and retina are rare. Ocular complications of malaria are uncommon considering the large numbers affected with this disease. These are vascular disorders affecting the retina, choroid and nerve fibres. It is difficult to assess the importance of cataract as a cause of blindness. It is more common and occurs at an earlier age than in Europe. The intracapsular extraction of cataract is the operation advocated by the authors.

E. O'G. Kirwan

TSOPELLAS, V. **Lutazol and Ophtazol in the Treatment of Trachoma.** *Chinese Med. J.* Shanghai. 1949, Oct., v. 67, No. 10, 551–4.

The treatment of 48 cases of trachoma by means of the sulphonamide known as G33 is discussed. Lutazol and Ophtazol, or G33 are manufactured by the Roussel Laboratories. Lutazol is the sodium salt of parasulphamidophenylazosalicylic acid. Ophtazol is the lithium salt of the same acid.

Lutazol is used in the form of ampoules containing 2 cc. of a 2 per cent. injectable solution or in tablets of 0·50 gm. Ophtazol is used as a 1 per cent. collyrium solution.

There are two successive steps in administering the drug, an initial phase (intensive treatment) and a complementary phase. The intensive treatment used by the author consisted in giving 12 tablets during the first two days, 10 during the third, 8 during the fourth and fifth days (50 in all). At the same time, 2 drops of Ophtazol were instilled into the eyes 3–5 times a day. Finally, 2 cc. of Lutazol were injected subconjunctivally on the first and eighth days of treatment. The complementary phase consisted in giving 4–8 tablets daily for 2–3 weeks after the initial treatment. The same amount can be used after a respite of 2–3 weeks.

The author formed the following conclusions :—

Treatment of trachoma by means of G33 has proved effective. In complicated cases of trachoma with corneal ulcerations and pannus, it has produced excellent results. It has proved adequate in those forms of trachoma with secondary infections. It does not, however, radically produce a cure of trachoma leading to the complete disappearance of trachomatous follicles and must therefore be combined with diathermic cauterization for the eradication of these follicles. G33 was found to be the most easily tolerated of all the sulphonamides and no dangerous subsidiary after-effects have been observed.

E. O'G. Kirwan

MISCELLANEOUS DISEASES

COLONIAL OFFICE. **Studies in Mental Illness in the Gold Coast** [TOOTH, G.]. Colonial Research Publications No. 6. pp. iv+72. 1950. London : H.M. Stationery Office. [2*s.* 6*d.*]

Dr. Tooth has carried out three investigations in the Gold Coast during a period of two years. The first concerned juvenile delinquency, and the second and third, which are reported here, the mental effects of trypanosomiasis and the forms taken by mental illness.

The mental effects of trypanosomiasis.—Trypanosomiasis is one of the commonest causes of mental derangement in the Gold Coast. A good deal is known of the physical effects and pathology of the disease, but diagnosis is sometimes delayed, because the significance of the early signs of mental disturbance is not fully appreciated. Abnormal sleeping may be a comparatively late sign. Accordingly, Dr. Tooth has paid attention to the minor mental symptoms especially. The central nervous system is invaded in the second stage of the disease, and there is then a meningeal reaction, but rarely meningismus or signs of raised intracranial pressure. Mental symptoms become evident during the third stage when the brain substance becomes involved. They are then not unlike those associated with cerebral forms of syphilis.

The minor mental symptoms fall into four categories : changes of personality or behaviour, with disorders of judgment especially ; affective disorders, with

the so-called " silent grief " or, less often, hyperactivity and euphoria ; intellectual impairment ; habit dilapidation, hallucinations, dreams and hypnogogic states. These changes were found in over four-fifths of a series of early cases ; and in two-fifths of the patients in whom abnormal sleeping was not present. The disease is sometimes confused with schizophrenia when laboratory investigations cannot be carried out.

Mental illness.—There is only one institution in the Gold Coast for the care of the insane, and only a very small proportion of the mentally ill are admitted to it. The large majority are cared for at home, usually under conditions which compare favourably with any that could readily be provided for them by the European authorities. There are good reasons for developing a mental health service in such a way as to continue this tradition, for institutional care is necessary only for a small number of illiterates and chronic literates who show anti-social and possibly dangerous tendencies ; the mental hospital should concentrate on the treatment of acute cases ; the majority of such patients will probably be literate.

Dr. Tooth tried to overcome the considerable difficulties of ascertainment, but was unable to do so. He was able to examine a sufficient number of patients, however, to reach some conclusions on the main stresses responsible for mental illness and the predominant types of disorder. The incidence of lunacy in sample areas of the Northern Territories was found to be between 96 and 60 per 100,000, but this was probably an underestimate. Rapid social evolution was thought to be one factor responsible for disease, although there was no evidence that Europeanization was a significant cause. Exposure to disease was another important factor, especially parasitic infections. Delusional psychosis was a common form of disease. Schizophrenia was found to take a similar form in literate Africans as in Europeans. The delusional content varied with the cultural background. Affective states were found to be common, but simple depressive reactions were relatively uncommon.

D. Russell Davis

Denoix, P. F. Le cancer en Afrique Occidentale Française. Récapitulation de dix années d'études (1940–1949) (d'après les documents recueillis par le service de santé militaire). [**Cancer in French West Africa : Cumulative Results of Ten Years Enquiry (1940–1949).**] *Bull. Inst. Nat. Hyg.* Paris. 1950, July–Sept., v. 5, No. 3, 372–7.

This report is based on official medical records from French West Africa during the 10 years 1940–1949. In 60 per cent. of cases the diagnosis of malignancy was confirmed histologically.

The results are shown in three tables, indicating respectively the number of cases registered each year in each of the 9 States, the cases by site and sex and the numbers by age-groups and sex. There is also included a reproduction of a model form used for collecting the records.

The total number of cases notified has increased to sixfold (from 68 in 1940 to 405 in 1949). This is attributed largely to increased recognition and reporting. The population of each of the states is shown : this varies from less than 400,000 in Mauritania to over 4 million in the Ivory Coast : the total population of French West Africa is approximately 16½ million. The average incidence of cancer for 1949 is given provisionally as 2·5 per 100,000 of the total population, varying from 1·3 per 100,000 in Dahomy to 11 per 100,000 in Senegal.

The author believes that malignant disease is no less common in Africans than in Europeans. Unfortunately the inevitably inadequate statistics available among primitive peoples do not permit of the most valuable comparison, namely by age-groups. It is hoped that increasingly regular statistics will

make it possible to obtain a more accurate picture of cancer in Africans and a comparison with its incidence in Europeans.

From the tables it is noted that 1,658 cases altogether were registered during the 10 years. In 986 the site was known exactly. Attention is drawn to the frequency of primary carcinoma of the liver (39 per cent. in men and 10 per cent. in women) and to cancer of the breast (28 per cent.) and of the uterus (22·5 per cent.) in women. The ratio between epithelioma and sarcoma was 82 : 18 in men and 92 : 8 in women.

The table showing incidence by sex and age-group is admittedly very approximate, owing to the difficulty in estimating the age of Africans.

This report forms a valuable addition to the knowledge of cancer incidence in tropical Africa. *H. J. O'D. Burke-Gaffney*

HARTZ, P. H. **The Incidence of Sarcoma, Leucaemia and Allied Diseases in the Native Population of Curaçao, N.W.I.** *Documenta Neerlandica et Indonesica de Morbis Tropicis.* Amsterdam. 1950. June, v. 2, No. 2, 161–5. [23 refs.]

"Of a series of 2,200 autopsies performed in Curaçao, N.W.I., 1,973 were performed on indigenous (almost exclusively coloured) people. Among these 1,973, 202 carcinomata and 27 sarcomata were found. Leucaemia was found in 9 cases. Hodgkin's disease in 3. These are compared with those from North and Central Africa. Some hypotheses which try to explain the differences in frequency of various tumours (especially of the tumours of the so-called reticulo-endothelial tissue) are briefly discussed."

TELLES, W. Síndrome pulmonar eosinofílica. **The Eosinophilic Lung Syndrome.** 79 pp., 17 figs. [Bibliography.] 1948. Rio de Janeiro : Avenida Graca Aranha, 226.

This is a thesis presented to the National Faculty of Medicine, Rio de Janeiro, and dealing with the subject of eosinophilic lung in 11 brief sections and a fairly full bibliography. It is in the main a summary of what we already know, but it is useful to have such summaries of obscure conditions from time to time.

In an introductory section the author divides cases into four types : 1. The typical Loeffler syndrome, transient and benign, known also as the Leitner syndrome [though Leitner's account appeared 5 years or so after Loeffler's]. 2. Severer, septic forms of longer duration. 3. Benign but protracted, clearing up spontaneously. 4. "Tropical eosinophilia" or "eosinophilic pneumopathy cured by arsenic".

Section 2, on the incidence of the condition, gives a few figures of frequency recorded from different parts of the globe. Section 3, on the clinical aspect, describes the so-called typical case, with illustrative radiographs of the thorax, and the more protracted cases ; the severe chronic type which the author calls the Lohr type ; the mild chronic or Bass type, and the tropical eosinophilia of Frimödt-Moller and others. Next, a little more is said on the radiological pictures and 5 types are mentioned : i. Extensive shadows of irregular contour in one or both lungs ; ii. Nodular opacities ; iii. Multiple small shadows, unilateral or bilateral ; iv. Sharply defined lobar infiltrations ; v. Wedge-shaped shadows sometimes extending to the hilum. Haematology, the next section, is disposed of in just over a page and then follows four pages on the pathological anatomy. This has little value since the condition is not fatal and the lesions found might equally be due to the actual cause of death and not to any concomitant lung infiltration and eosinophilia. A most important section follows, namely on

the *Aetiology*, and the various ascriptions are referred to and briefly discussed, under the heads of *microbial*, a transient pneumonia, brucellosis, or the inevitable hypothetic virus ; *parasitic*, association with *Ascaris* larvae (with illustrative cases), larva migrans, *Dirofilaria immitis* and *Fasciola hepatica* ; *allergy* to bacteria or parasites, or to food, drugs, etc. For General Diagnosis the chief features are transient lung shadows the evolution of which can be followed in a series of radiographs, a leucocytosis with eosinophilia of varying degrees ; general systemic manifestations, usually mild, an allergic history "nearly always obtainable" and, lastly, a favourable response to arsenical treatment in those coming under the head of "tropical eosinophilia". Differential diagnosis has to be made from [this is the author's grouping] : other allergies, dermatoses, parasitic infestations, infections and inflammation, the effect of drugs and from "local eosinophilia". Evolution and progress are disposed of in a short section of half a page. Treatment is indicated as dependent on the nature of the allergy and the presence of parasites and, in the tropical eosinophilia form, the giving of arsenic. Finally, there is a bibliography of more than 200 references showing that the author has consulted the literature of the subject widely. *H. Harold Scott*

Thompson, B. **South-West Pacific Pulmonary Disease.** *J. Trop. Med. & Hyg.* 1950, Jan., v. 53, No. 1, 1–5. [22 refs.]

In the course of a study of pulmonary tuberculosis in Western Samoa, the author was impressed by the number of cases that exhibited a radiological picture that would be considered abnormal on British and American standards. The radiological picture in the mildest forms showed bilateral heaviness of the bronchial markings, increasing towards the hilum, with perhaps some obscuration of the costophrenic sulci. In the more advanced stages, there was definite fibrosis in one or more zones radiating from the hilum with contraction of the vessel markings and perhaps gross pleural thickening ; this was usually more marked on one side, and often led to mediastinal displacement. There was no calcification or cavity formation.

In a study of 167 hospital cases, diagnosed as pulmonary tuberculosis between 1944 and 1948, the diagnosis was confirmed in 109 [119 ?] cases by the presence of tubercle bacilli or of unequivocal radiological evidence, but in the remaining 48 it was rejected, although the symptoms had been typically those of phthisis.

In a group of Samoan girls, candidates for enrolment as nurses, many instances of the above type of interstitial fibrosis were found ; some of these had been rejected on this account.

In a fluoroscopy study in a village, 7 of 292 persons were selected and the abnormality confirmed by skiagrams. This showed the radiological picture described above. Three had already been diagnosed clinically : they all had a productive cough and shortness of breath, and gave a long history of symptoms including the frequent spitting of blood. In none of these were tubercle bacilli found even on culture ; nor was there evidence of bronchiectasis.

In a group of 40 persons, poor European or partly Samoan, who were selected on account of a high eosinophilia, 14 showed excess lung markings of which 6 were considered definitely fibrotic ; two showed nodular foci.

The possible aetiology is discussed. Both intestinal helminthiasis and filariasis are very common in Samoa. Eosinophilia is almost constant ; in a group of 91 "healthy" schoolboys, over 20 per cent. of eosinophiles was found in 39, and over 10 per cent. in 74. The author considers that the condition is different from either Loeffler's syndrome or tropical eosinophilia, and that the most likely cause of the interstitial fibrosis is the frequent migrations of intestinal worms through the lungs. He adds, however, "filariasis may also play a part". *L. E. Napier*

ROBERTS, M. **Treatment of Thrombo-Angeitis Obliterans by Hypoglycaemia.** *East African Med. J.* 1950, July, v. 27, No. 7, 289–90.

In this paper, the author describes the effect of hypoglycaemia induced by insulin in a case of obliterative vascular disease of the lower extremities, conforming to the clinical picture of thrombo-angeitis obliterans, which he believes to be a not uncommon disease in Africans.

The patient was an African male from Nyasaland, aged 34, who was admitted to hospital in September 1946 with gangrene in the big toe of the right foot. The terminal phalanx of this toe was amputated. However, spreading gangrene set in later and a mid-thigh amputation followed in May 1948. In September of the following year, gangrene developed in some of the toes of the left foot. It spread steadily. A retroperitoneal lumbar sympathectomy was performed, but resulted in no improvement. Further excision of the gangrenous tissue had to be carried out, but the patient was left with a deep, painful ulcerating cleft between the 1st and 5th metatarsals.

Early in December 1949, he was given a first 20-day course of insulin (20 units at 7 a.m. and 30 units at 2 p.m., after meals). From about the third day he stated that the foot felt warmer and the pain was much relieved. Several days later improvement in the local condition was evident. The gangrene had spread no further and the ulcerated area was now covered with clean red granulation. On the twentieth day, the site was ready for skin grafting, which was then done.

In early February 1950, a second course of insulin treatment was instituted, 20 units being administered twice a day. Healing was soon complete. The patient was discharged from hospital at the end of the month with a soundly healed foot stump and with the fifth toe intact.

The treatment, originally described by MAZANEK (*Lancet*, 1949, Nov. 19, 935) is based on the theory that thrombo-angeitis is caused by a trophic disturbance of the vessel walls, which is relieved by insulin. *M. Gelfand*

BENDER, P. B. **Ainhum. Report of a Case with Bilateral Involvement.** *U.S. Armed Forces Med. J.* 1950, Sept., v. 1, No. 9, 1039–43, 1 fig. [22 refs.]

A case in an American Negro.

BUTZ, W. C. & ELTON, N. W. **Report of Two Additional Cases and an Analysis of the Pathogenesis of Ainhum on the Isthmus of Panama.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1950, July, v. 44, No. 1, 113–16, 6 figs. [13 refs.]

PHILLIPS, P. & MURPHY, M. A. **Tick Paralysis.** *Canadian Med. Ass. J.* 1950, July, v. 63, No. 1, 38–9.

Two cases in children seen in British Columbia.

PROTOZOOLOGY : GENERAL

RODHAIN, J. Formation de pseudokystes au cours d'essais d'immunité croisée entre souches différentes de toxoplasmes. **Pseudocysts formed in the Course of Cross-Immunity Tests with Different Strains of Toxoplasms.** *C. R. Soc. Biol.* 1950, May, v. 144, Nos. 9/10, 719–22, 1 fig.

As there is still some doubt regarding the unity or plurality of species of *Toxoplasma*, a question which has not been adequately settled by cross-immunity tests, the author has carried out a number of such experiments

in an attempt to throw further light on this problem. For this purpose 2 strains were used, both maintained in mice. The first (S.H.) was a human strain from Holland, which produced an acute infection terminating in death within 8 days. The second (E.III), isolated from a squirrel in Antwerp, produced mild chronic infections, sometimes lasting several months.

The cross-immunity tests were carried out by inoculating the human strain of toxoplasms into mice infected with the squirrel strain. In all cases (except one which was doubtful), infection with the squirrel strain partially protected the mice against superinfection with the human strain, since it prevented the development of acute symptoms usually produced by the latter but did not inhibit the development of the parasites. These were present in various organs, especially in the brain, where pseudocysts were found. The identity of the human strain in the superinfected mice was determined by inoculating small quantities of their brain emulsion into clean mice. The resulting infections were of the acute type characteristic of the human strain.

In the course of these experiments it was noted that the pseudocysts, which were present in the brain and which probably represent the resistant stages during the latent period of infection, remained intact after the cerebral tissue was teased up with glass beads. In the emulsion they were freed of the host-tissues and appeared in the form of rounded bodies (diameter from 14·5 to 37·7 μ) packed with the refringent parasites and enclosed in a thin double-contoured membrane, as shown in an accompanying photomicrograph. On account of their individuality and resistance to mechanical shock, the author regards these structures as true cysts, rather than " pseudocysts ".

C. A. Hoare

KEMP, Gisela. Zur Klinik und Epidemiologie der Toxoplasmose. [**Symptoms and Epidemiology of Toxoplasmosis.**] *Klin. Woch.* 1950, Sept. 15, v. 28, Nos. 35/36, 602–6, 3 figs. [16 refs.]

The author speaks of three different types of toxoplasmosis, the infantile, the juvenile and the adult, and a fourth group, chronic, mild or symptomless and recognizable only by the serum tests. The chief symptoms are internal hydrocephalus or microcephaly—the two may be combined—with cerebrospinal fluid changes such as xanthochromia, cell count, high albumin and pathological colloid curves ; cramps, spasms, rigidity, tremor, athetosis, paralyses, ocular disturbances, chorio-retinitis, uveitis, optic atrophy, nystagmus, reduced field of vision ; and foci of calicification in the brain.

Two cases are described in detail. In one, the symptoms appeared before the age of 4 months, but signs were noticed soon after birth, such as apathy, torticollis and " disturbances of sight ". At the age of 9 months symptoms of acute encephalitis supervened, but the cerebrospinal fluid contained an excess of albumin without cell changes ; the final onset of stupor led to a fatal issue in 18 days.

The second was a child of 3 months, the last of a large family which included 7 full-time births, 4 early (6 to 7 months) and 3 miscarriages ; the mother, the 3 eldest daughters and the youngest, the patient, gave a positive Sabin serum test. The child's head was large, especially in the occipital region and there were present choroiditis, nystagmus, muscular rigidity and signs of internal hydrocephalus ; the antibody neutralization test was strongly positive. At the age of 4 months the child had an attack of whooping cough, became steadily weaker and died at the age of 6½ months.

To sum up : both children showed three of the four symptoms described by Sabin as characteristic of toxoplasmosis (all but the calcium deposits in the brain). The second gave a positive neutralization test during life and the

diagnosis was confirmed in both *post mortem*. One of the sisters with toxoplasmosis was largely concerned with nursing the second patient and the author suggests the possibility of direct person to person infection, but the fact of one of the parents and three sisters reacting positively and the many miscarriages and premature births, if ascribable to the toxoplasmosis, are evidence of other possibilities of infection. *H. Harold Scott*

Verlinde, J. D., Makstenieks, O., Beekman, H. & Copper, A. C. Clinische, parasitologische en serologische waarnemingen in het beloop van congenitale toxoplasmosis. [**Clinical, Parasitological and Serological Observations in a Case of Congenital Toxoplasmosis.**] *Nederl. Tijdschr. v. Geneesk.* 1950, Sept. 23, v. 94 (iii), No. 38, 2746–54.

The English summary appended to the paper is as follows :—

" Description of a case, clinically, parasitologically and serologically controlled during the first five months, of an infant suffering from congenital toxoplasmosis with hydrocephalus, lime foci in the brain, microphthalmus and anomalies in the fundus oculi. At different points of time during the first three months, a toxoplasma strain (vB) was isolated from the ventricular liquor and from the blood. By means of the staining and complement-fixation test, a gradually increasing quantity of anti-bodies was demonstrated in this patient's serum and in his mother's. The anatomical eye anomalies as well as the results of the serological test might point to infection of the infant, perhaps also of the mother, during the last two months of gravidity. In spite of treatment of the patient with percoccide from the 19th to the 36th day and in spite of the anti-body titre, which in the meantime already had mounted very high, during and after this period living parasites could still be demonstrated in blood and cerebrospinal fluid."

Mohr, W. & Westphal, A. Zur Klinik und Therapie der Toxoplasmose. (Vorläufige Mitteilung.) [**Toxoplasmosis and its Treatment (Preliminary Communication).**] *Med. Klin.* 1950, Sept. 15, v. 45, No. 37, 1167–8.

Many points, say the authors, in the symptoms, diagnosis, epidemiology and treatment of toxoplasmosis are still obscure. They go on to discuss the Sabin-Feldman test [this *Bulletin*, 1950, v. 47, 496] and the effect of aureomycin on it. Those with a positive reaction before the treatment showed a change of titre after it. Further, they observed two patients who gave a positive result though they showed no symptoms. This led them to consider the effect of aureomycin on possible latent toxoplasmosis. They divided their patients into 5 groups :—

1. Women with a positive Sabin-Feldman (S.F.) test, whose children certainly had toxoplasma infection (determined in some cases by autopsy).
2. Men with cardiovascular lesions, and giving a positive S.F. test.
3. Those with *Leptospira canicola* infection, some of whom react positively.
4. Patients with fever and ill-defined and undiagnosed clinical conditions, who gave a positive reaction.
5. Those having experimental laboratory infections with *Toxoplasma*.

The dosage of aureomycin was 500 mgm. orally every 6 hours for two or three days, *i.e.*, 2 gm. daily and 4 to 6 gm. in all ; if given intravenously 100 mgm. every 6 hours. All groups showed a fall in titre in 2 to 4 weeks after the treatment and Westphal believes that the aureomycin destroys the parasite.

Clinical features vary with the age of the patient ; in children encephalitic symptoms are common ; in adults pneumonic or enterocolitis. In animals the last is the most often seen.

Following up the work of Westphal the authors tested a number of patients with obscure febrile conditions by the S.F. test and group them thus :—

1. Those with fever and cerebral symptoms of an epileptiform character (2 cases).
2. Those with fever and myocardial signs (1 case).
3. Those with enteritis and fever. One patient had had these symptoms before giving birth to a dead hydrocephalic infant.
4. Those with fever of a septic type for 2 to 3 weeks with asthenia and anaemia, but no discovered cause (1 case).
5. A case of relapsing pneumonia with pleural involvement, persisting on and off for a year. Patients of this type were given a larger dosage of 5–8 gm. of aureomycin.

All these gave a positive S.F. test and on the possibility that they might be cases of toxoplasmosis all were given aureomycin.

With so few cases it is not surprising that no definite conclusions could be reached. [It will be noted that this is a preliminary communication.] The stages in the investigation will be, first, to determine whether the test is specific or not, and then to find out what proportion of the various undiagnosed maladies with fever are due to toxoplasmosis and what diseases give a non-specific reaction. If the test proves to be specific it will be most serviceable in the diagnosis of cases in an early stage before irreparable harm is done.

H. Harold Scott

Freeman, P. & Pryor, Helen B. **Toxoplasmosis in a Nine-Year-Old Girl.** *J. Pediatrics.* St. Louis. 1950, Mar., v. 36, No. 3, 365–9, 4 figs. [14 refs.]

Jacobs, L. & Jones, Frances E. **The Parasitemia in Experimental Toxoplasmosis.** *J. Infect. Dis.* 1950, July–Aug., v. 87, No. 1, 78–89. [14 refs.]

This is a careful study of the extent to which *Toxoplasma* invades the blood stream of infected animals and it should supply valuable data in the search for a possible arthropod vector of the disease. Two strains of the organism were used, human and canine, chiefly the former. Animals were given measured amounts of infective material obtained by diluting the peritoneal exudate of mice infected for 3 or 4 days. Then blood was taken from the subinoculated animals on different days after infection and injected in various dilutions into receptor mice. The latter were killed after a week, and brain, liver and spleen smears were examined for the organisms, and other mice were inoculated with the ground-up organs. Mice, rats, rabbits, pigeons and chickens were tested in this way and parasitaemia was observed in all. In mice which had been given a heavy dose, the blood became infected the next day ; and in almost all cases the blood was constantly infected for 4 or 5 days before the death of the animal. In chronic infections, parasitaemia persisted irregularly for three months. The disease is less acute in rats and the organisms could be demonstrated only occasionally in the blood. In rabbits, parasitaemia developed on the second or third day after a heavy dose, and by the fourth day, after a smaller dose. In pigeons, parasites appeared in the blood 4 to 7 days after inoculation, persisted for 1 or 2 weeks and then disappeared abruptly. In pigeons with a latent infection, parasitaemia was invariably present during the second week after infection and might occur also at later dates. The infection in chickens ran a somewhat similar course to that in pigeons though the parasitaemia in chronic infections was less constant. The number of parasites found in any of these animals with the exception of rats was approximately of the same order regardless of the route of inoculation. In rats, 0·5 cc. of dilutions over 1 : 3 were apparently uninfective. In the other animals,

an increasing degree of parasitaemia developed as the infection progressed; infection resulting from 1 : 10 dilutions to 1 : 1,000, and even up to a maximum dilution of 1 : 5,000. This last was thought to be equivalent to about 50 organisms per cmm. of blood. Blood smears of rabbits showing a high parasitaemia were searched and in 2 cases a single organism, lying free, was found after several hours. Subsidiary experiments showed that *Toxoplasma* was consistently absent from skin scrapings of infected animals in the absence of parasitaemia. The authors suggest that arthropods which feed on both avian and mammalian hosts should be studied as possible vectors of *Toxoplasma*.

P. C. C. Garnham

Rodhain, J. La grande réceptivité des Sciuridés aux toxoplasmes. [**Susceptibility of Squirrels to Infection with Toxoplasms.**] *C. R. Soc. Biol.* 1950, May, v. 144, Nos. 9/10, 717–19.

The author has previously recorded a case of spontaneous toxoplasmosis in a marmot at the Antwerp Zoo. He subsequently found three infected squirrels (*Sciurus vulgaris*) which lived in the same cage as the marmot and died of toxoplasmosis. In these animals various organs showed pathological changes and contained parasites, which were especially numerous in the liver. Inoculation of material from the squirrels to mice produced mild chronic infections. The peritoneal fluid of a 3rd passage mouse was inoculated intraperitoneally into a squirrel, which died 8 days later. Autopsy revealed numerous toxoplasms in the liver, a fair number in the lungs and a few in the spleen and brain. From observations on the four squirrels it is concluded that these animals are highly susceptible to toxoplasmosis.

The author recalls that within the family Sciuridae the following species have been found to be susceptible to infection with *Toxoplasma* : *Marmota arctomys marmota, Sciurus vulgaris, Citellus pygmaeus, C. fulvus* and *Xerus getullus*.

C. A. Hoare

ENTOMOLOGY AND INSECTICIDES : GENERAL

Oldroyd, H. **Diptera. I. Introduction and Key to Families.** *Handbooks for the Identification of British Insects.* Roy. Entom. Soc. London. 1949, July 29, v. 9, Pt. 1, 49 pp., 97 figs. & 1 coloured pl. [52 refs.] [7*s.* 6*d.*]

Among the new series on British insects published by the Royal Entomological Society of London there has recently appeared this useful introductory booklet on the Diptera. A very clear account is given of the structures used in the systematics of the order, with copious illustrations, which supplement the keys to the families. Only the adults are treated in detail but figures of representative larvae and pupae are included. The booklet constitutes an up-to-date and practical introduction to the subject and should also be of value to those whose interests embrace a wider field than the fauna of the British Isles.

M. T. Gillies

Rempel, J. G. **A Guide to the Mosquito Larvae of Western Canada.** *Canadian J. Res.* Sect. D. Zool. Sci. 1950, Aug., v. 28, No. 4, 207–48, 1 map & 43 figs. [15 refs.]

" Keys to the genera and species of mosquito larvae of the four western provinces of Canada are presented. To facilitate identification the generic and specific characters used in the keys are fully illustrated. A map of Western Canada showing the main vegetation zones and the principal areas of intensive mosquito collecting is included. Six genera and 46 species are represented in

the mosquito fauna of Western Canada, namely *Anopheles* with four species, *Mansonia* with one species, *Wyeomyia* with one species, *Culiseta* with five species, *Culex* with four species, and *Aedes* with 31 species."

HADDOW, A. J., GILLETT, J. D., MAHAFFY, A. F. & HIGHTON, R. B. **Observations on the Biting-Habits of some Tabanidae in Uganda, with special reference to Arboreal and Nocturnal Activity.** *Bull. Entom. Res.* 1950, May, v. 41, Pt. 1, 209–21, 3 figs. [11 refs.]

An account is given of Tabanids taken in the forests of Uganda in the course of investigations into the vectors of yellow fever. *Chrysops centurionis*, the principal species, was found to be mainly arboreal and, in its biting activity, crepuscular.

Work was carried out on platforms in the forest canopy and at lower levels, with African boys acting as bait. In the wet season *C. centurionis* showed a very marked preference for the canopy at the 58 and 82-foot levels. In the dry season it was taken much lower, mostly between 20 and 30 feet but a certain number were found at all levels. In the wetter type of forest at Mongiro the level during the dry season was rather more well-defined at the 31-foot level. This species evidently prefers a good deal of shelter and for this reason avoids the sparse foliage of the canopy in the dry season.

The main biting activity of *C. centurionis* was in the hour before and the two hours after sunset. Some activity, however, continued right through the night. This behaviour closely resembles that of *Aëdes africanus*, a proven vector of jungle yellow fever, and an inhabitant of the canopy. It is suggested that these two unrelated insects may be controlled by the same factors and that their natural food preferences, *i.e.*, monkeys, may also be the same. In this connexion it is thought possible that *C. centurionis* may be the vector of certain filarial infections in monkeys.

Five other Tabanids were also noted to be nocturnal in habit, one species of *Tabanus*, three of *Haematopota* and another species of *Chrysops*. Records of other species of Tabanids from Bwamba are given. *M. T. Gillies*

DAMASCENO, R. G., AROUCK, Regina & CAUSEY, O. R. Estudos sôbre Flebotomus no vale Amazônico. Parte VI. Contribuição ao conhecimento da distribuição geográfica e da incidência por tipo de captura, de 64 espécies identificadas. [**Studies of *Phlebotomus* in the Amazon Valley, Part VI. Geographical Distribution and Incidence by Type of 64 Species Identified.**] *Rev. Serviço Especial Saúde Pública.* Rio de Janeiro. 1949, July, v. 2, No. 3, 817–42, 65 maps on 4 pls. [27 refs.]

The English summary appended to the paper is as follows :—

" The authors describe the geographic distribution by municipality and the incidence by type of capture of 64 species of *Phlebotomus* (Diptera, Psychodidae) studied in the States of Amazonas and Pará, Brazil, from August 1942 to April 1945."

PEUS, F. Die Flöhe. Bau, Kennzeichen und Lebensweise, Hygienische Bedeutung und Bekämpfung der für den Menschen wichtigen Floh-Arten. [**Structure, Identification and Biology, Hygienic Importance and Control of Species of Fleas important to Mankind.**] *Hyg. Zool.* 1938, v. 5, 106 pp., 29 figs. [107 refs.] Leipzig : P. Schöps.

The author deals with the structure, identification, hygienic importance and control of species of flea important to man and domestic animals. Emphasi

is laid on the biology of fleas where it is a guide to methods for their control. The monograph is divided into 4 parts. Part I, of 46 pages, deals with structure, systematics and development. Except for the reproductive system, internal anatomy is rarely mentioned. There are keys to adult and larval stages of fleas of economic importance in Europe. The keys are excellently illustrated with bold and clear drawings. In just over 6 pages 19 species are summarized in respect of host-relationship and breeding range.

Part II deals with the habits of adult, larva and pupa and there is a short account of oviposition and development. Adults are considered at greatest length : their discussion occupies 15 pages. There are paragraphs on host relationship, distribution, sensory physiology, locomotion and dissemination, feeding habits and propagation.

Four and a half pages are then devoted to the effect of environmental factors on the duration of each stage. Temperature and humidity are principally mentioned. This is followed by a short and interesting section on the life cycle and longevity of different species of flea in relation to the habits of their hosts. There are 3 pages on parasites of fleas : these parasites are mainly protozoa. Bacterial symbionts are mentioned and there is a section on enemies of fleas. In this section the author considers factors such as predators and weather which keep numbers of fleas in check. An appendix of 7 pages follows. This contains useful notes on means of collecting, preparing, breeding and keeping fleas.

Part III is entitled " Hygienic Importance of Fleas" and in its 9 pages the author mentions the " nuisance value " of different fleas to man and domestic animals in Germany. Dog fleas may be a great nuisance to man. The author considers the effects of bites on people and also fleas as intermediate hosts of two flatworms. The dog flatworm *Dipylidium caninum* and the rat flatworm *Hymenolepis diminuta* can be transmitted to man. The method of transmission is outlined. Five pages are devoted to the transmission of diseases in warm climates. The author mentions fleas as possible carriers of filarial worms, Leishmania and Bartonella : also the survival of bacteria such as *Staphylococcus aureus* occurs in the larval flea till emergence when it can be transmitted by the feeding of the adult flea. The author thinks rat and mouse fleas may carry typhoid fever. He also describes infection experiments with pneumococci. Plague is mentioned and the author warns us that plague can become established in places where central heating is maintained in buildings.

Part IV on Prevention and Control occupies 9 pages and is much out of date for the monograph was published before the new insecticides were widely used. There is a paragraph on the treatment of flea bites. *A. Smith*

Applewhite, K. H. & Smith, C. N. **Field Tests with Mosquito and Sand Fly Repellents in Alaska.** *J. Econom. Entom.* 1950, June, v. 43, No. 3, 353–7.

The authors describe tests on repellents against mosquitoes (*Aëdes*) and midges (*Culicoides*) carried out under natural conditions during summer in Alaska. The substances tested have, it is thought, been already selected by laboratory tests from a much larger number of compounds and have been examined for toxicity to man. The substances were tested as 25 per cent. solutions in alcohol on the arm. A number of different volunteers were used and in each case the two arms were used to give a direct comparison between two substances. Protection is measured as the number of minutes elapsing till the first " confirmed bite ", *i.e.*, the first bite, provided a second one occurred within 30 minutes.

One notices the large disparities that are met with between successive experiments : for instance propyl N, N-diethylsuccinamate was tested on nine men in one place, and gave periods of protection ranging from nine to over

334 minutes. It seems questionable whether any conclusion can be based on the first confirmed bite with such ranges of results as these, or whether any importance can be attached to the average period of protection. It is evident that the statistical problem is an extremely difficult one. In addition several species of *Aëdes* occurred in the area and in many of them the abstracter believes that the females are most difficult to determine, so that there may have been a large, undetected, biological variable.

The conclusion is reached that against the prevalent mosquitoes the substance mentioned above and dimethyl phthalate are not very different in their effectiveness and that both are much more effective than the other substances tested.

In other experiments repellent substances were applied to cloth and observations made on the protective effect of this against the bites of the prevalent mosquitoes. The most effective of the substances tested was hexyl mandelate, which remained effective for long periods, even after many rinsings with water.

Against midges (*Culicoides*) the materials were tested by application to gauze head nets. None of the substances gave adequate protection though in all cases the treated net gave a considerable measure of protection as compared with the untreated control. *P. A. Buxton*

Jones, T. W. T. **On Testing Anti-Malarial Oils for Toxicity.** *East African Med. J.* 1950, July, v. 27, No. 7, 269–73, 2 diagrams.

In testing the toxicity of antimalarial oils to mosquito larvae in the laboratory it is notoriously difficult to obtain a complete oil film from edge to edge of the dishes used. The cause of an incomplete oil film is the presence of an " invisible film " at the commencement of the experiment, originating from the fatty acids contained in sweat left on the edges of the dish by fingering. The author discusses briefly the physical phenomena of surface tension, interfacial tension, spreading pressure and surface-active substances in their relation to this problem. He concludes that the spreading power of an oil is related to the interfacial tension between oil and water ; an increase in spreading power may be attained by the addition of surface-active substances which alter the interfacial tension.

In practice the fatty acids from sweat are eliminated by allowing boiling water to fill and overflow from the dishes to be used and from then on careful handling enables the introduction of larvae under a complete oil film by the routine method of a vertical glass tube in the centre of the dish.

R. Ford Tredre

Alessandrini, Maria E. **Residual DDT Content. A Rapid Method for the Detection and Determination of Small Quantities of DDT on Sprayed Surfaces.** *Bull. World Health Organization.* Geneva. 1950, v. 2, No. 4, 629–36.

A rapid and fairly sensitive colorimetric method is described, which may be easily applied in the field, to detect and determine small amounts of DDT on most varied types of surfaces such as mortar, wallpaper, leaves and water, which had been treated with the insecticide in different forms, for example in kerosene solution, aqueous emulsions, wettable powders or aerosols. The method is derived from that of Schecter, Soloway, Hayes and Haller, which has been simplified and modified. With the aid of a special colour scale a series of 10 determinations can be carried out in an hour with sufficient accuracy for the requirements of malaria control without the necessity of using a colorimeter.

The dust obtained by scraping, or the extract left on washing a small part of a treated surface with ether and evaporating off the solvent, is nitrated with a 1 : 1·5 mixture of concentrated sulphuric acid and fuming nitric acid, the nitrated product extracted with benzene, and treated with approximately normal alcoholic caustic potash. A coloration develops which varies from pale to dark blue and the solution is transferred to a tube of standard dimensions, and the coloration compared with a colour-scale from which the amount of DDT originally present can be read off directly in milligrammes. A small number of copies of the colour scale are available from the Malaria Section, W.H.O., Geneva. A coloration is even obtained with 2 μgm. of DDT, which is faint but still distinct ; however, the colorations most suitable for comparison are those obtained with amounts of DDT varying between 0·0001 and 0·001 gm.

It is possible by this method to detect if the DDT is completely or partly decomposed. Thus, non-dehydrochlorinated DDT gives a blue coloration and within 24 hours a yellow coloration, whereas dehydrochlorinated DDT gives only a yellow coloration. The presence of chlordane modifies the blue coloration characteristic of DDT ; the coloration becomes weaker as the amount of chlordane increases. Consequently the method gives only qualitative results when applied to mixtures of DDT and chlordane.

The necessary apparatus consists of : a separating funnel of about 25 ml. capacity ; some dry test tubes ; some 5 ml. graduated cylinders ; small calibrated glass tubes with an internal diameter of exactly 7·2 mm. and about 10 cm. long ; a spirit lamp ; a beaker or any other vessel suitable for use as a water bath ; a test-tube rack ; and the special colour-scale on paper.

T. E. Fletcher

Smith, H. F., Dy, F. J. & Del Rosario, F. G. **The Use of the United States Army M-2 Smoke Generator for Mosquito and Fly Control.** Reprinted from *Acta Med. Philippina.* 1950, Jan.–Mar., v. 6, No. 3, 207–17, 2 figs.

In the City of Manila, Philippines, in 1948, trials were conducted to determine the efficiency of a modified United States Army M-2 smoke generator for fogging buildings and open spaces with insecticide to kill flies and mosquitoes. In assessment of results it is not easy to ascertain what proportion of flying insects escape to clear air ; however, the exposure of live insects in gauze cages gives a fairly reliable indication of the lethal effect. Five per cent. DDT was ineffective but 10 per cent. DDT gave favourable results in the case of mosquitoes. In the public markets, which were teeming with flies, very good results were secured with chlordane and gammexane, in the case of flies and culicine mosquitoes, particularly " 1 per cent. gammexane " for a fogging period of 5 minutes.

The precise preparation of gammexane is not mentioned.

The T.I.F.A. insecticidal fog applicator has been found superior to the M-2 and has been adapted by the Malaria Control Division of the United States Public Health Service as standard equipment for this purpose. Disadvantages in the use of the M-2 smoke generator and hazards to operators working close to it are discussed.

R. Ford Tredre

BOOK REVIEWS

VON SCHMIDT, H. **Durch Insekten hervorgerufene Krankheiten. Ihre Hygiene und Klinik.** [Diseases caused by Insects. Their Hygienic and Clinical Aspects.] 277 pp., 123 figs. 1949. Stuttgart : Ferdinand Enke Verlag. [Unbound D.M.30 : Bound D.M.32.50.]

In his preface the author writes that he hopes his book may prove " a building stone in the knowledge house of medical entomology ", and that he has described not only the entomological aspects of his subject, but also the clinical and hygienic aspects, in so far as these relate to insect-borne diseases. In practice, however, the scope of the work extends far beyond these self-imposed limits and includes accounts of many of the protozoa and of some of the metazoa which are transmissible to man by arthropods. Not only is this the case, but in certain instances the accounts of the parasites transmitted are far more detailed than are the descriptions of their vectors. Thus, the account of the sandfly, including illustrations, is limited to one and a half pages, whereas the description of the pathogenic organisms it transmits occupies another six pages. This method of treatment has resulted, at least so it appears to the reviewer, in a lack of balance, as if the author was not certain which aspects of his subject were of most importance. Thus, the account, which is an excellent one, of the venomous arthropods occupies some 54 of the total 277 pages which make up the book, which seems an undue proportion of space to devote to a relatively unimportant aspect of medical entomology, particularly when one observes that the tsetse fly is dismissed in less than two pages and the house fly in less than half a page.

Although this paucity of entomological information, particularly as regards the recognition of important genera and species of arthropods, robs the work of much of its value as a textbook of medical entomology, nevertheless, it should prove of considerable value to practitioners who wish to consult a brief account of medically injurious insects and the diseases they transmit or the injuries they may produce. The author states that the majority of the original illustrations were lost by war damage, but he has replaced them by borrowing from various text books—particularly Martini's *Lehrbuch der Medizinischen Entomologie* [this *Bulletin*, 1948, v. 45, 376]. The work is well produced and contains an excellent index, but there are numerous misspellings of scientific words, such as " *Trombicula delhiensis* ", " *Spirochaeta cartera* ", " *Chrysops dimidiatus* ", " *Chrysops silaceus* "; while obsolete names, such as : " *Pediculus vestimentorum* ", " *Conorrhinus infestans* ", " *Microfilaria nocturna* ", " *Aedes fasciatus* ", " *Ceratopogon grahami* ", " *Ceratophyllus fasciatus* ", " *Filaria perstans* ", " *F. ozzardi* ", are frequently employed.

R. M. Gordon

SAWITZ, William G. [M. D.]. **Medical Parasitology. For Medical Students and Practicing Physicians.** pp. xi+296, 90 figs. (1 coloured). 1950. Philadelphia & Toronto : The Blakiston Company. [$4.25.]

The author states in his preface that his book is intended as a lecture and laboratory guide for students (sophomores) in their course in medical parasitology, that the subject is presented from the medical rather than from the zoological aspect, and that the book does not, and is not intended to, replace text-books essential for parasitologists. The publishers " insert " which accompanies the book quotes from the author's preface " but "—it adds—" it does provide the medical student with all the information he needs." Unquestionably the author has gathered together in a relatively small text-book

an astonishingly large amount of information concerning arthropods, helminths and protozoa which adversely affect mankind in temperate and tropical climates, and not only should the American student who has mastered its contents know far more than his counterpart in Britain, but he should require but little extra coaching, so far as parasitology is concerned, before passing his post-graduate diploma in tropical medicine. Unfortunately, however, this knowledge is presented in such a condensed form that the reader is left with the impression that it could only be assimilated by a prodigious feat of memory. Furthermore, it would appear probable that in the absence of a consecutive account of the life-cycle and habits of, at any rate, the more characteristic parasites and their vectors—nearly 200 are mentioned—he will never understand the implications and applications of what should be a fascinating subject, and will soon forget his carefully memorized facts. This over-emphasis on names and unexplained statements is particularly unfortunate since obviously Dr. Sawitz is well qualified to write on his subject and when he allows himself sufficient space, as in the section dealing with the life-cycle of the malaria parasite, he presents an interesting, adequate and clear-cut picture. Similarly in the section dealing with the treatment of parasitic infections and with the use of insecticides, in which section the author has collaborated with Dr. Harry Most, much more space is used, nearly one-quarter of the book, with the result that the presentation of the subject, although concise, is adequate and lucid.

It is in the systematic portion of the book, which occupies more than half the contents, that over-condensation is most apparent, and in addition there are a number of inaccurate statements together with certain omissions, which might well mislead a student. The following are quoted as examples : p. 35—writing of *T. gambiense* and *T. rhodesiense* " Both occur only in Central Africa ". p. 36—when describing the laboratory diagnosis of these infections reference is made to sternal puncture, but the examination of a fresh preparation made from peripheral blood is omitted. p. 55—referring to *P. falciparum* " sternal, splenic or placental blood must be examined to recover the stages older than six hours. If asexual stages older than six hours are found in peripheral blood, the prognosis is fatal." [The reviewer knows of no good evidence that older stages of *P. falciparum* are more numerous in the spleen and sternal marrow than in the peripheral blood ; while the statement regarding prognosis is apt to mislead.] p. 56—a key for the identification of plasmodia contains the statement : " Plasmodia, all of them in the film in the ring stage, are *P. falciparum.*" This may be good teaching for early treatment of the patient, nevertheless it is inaccurate. p. 94—in referring to *Wuchereria bancrofti*, the author writes : " Night-biting mosquitoes suck up these microfilariae. Those not taken up die within three weeks. . . . The important vectors are *Culex fatigans* (*quinquefasciatus*), a cosmopolitan night-biting mosquito, and *Aedes scutellaris*, a day-biting mosquito in the Pacific." What evidence exists suggests that the microfilariae can persist much longer than three weeks. The failure to mention anophelines as important vectors in the transmission of *W. bancrofti* is misleading. p. 111—it is stated of *T. saginata* that " It feeds on the food of the patient, resulting in a loss of weight when the food intake is not sufficient for both the patient and the worm " ; a similar statement concerning *T. solium* is made on p. 114. These beliefs are not in accordance with present-day teaching. p. 116—the description of the life-cycle of *Diphyllobothrium latum* concludes as follows : " The sparganum larva of *Diphyllobothrium* can infect man, causing sparganosis. Man becomes infected with the sparganum either by ingesting the procercoid larva in the crustacean or by using as a poultice flesh of fish containing the sparganum. Sparganosis in man is infrequent." [When used as poultices frogs—not fish—are usually regarded as responsible for the direct transmission of sparganosis to man.] p. 136—*Paragonimus*

westermani is described as " limited to the Far East ", whereas it also occurs in Africa. p. 141—to describe millipedes as " innocuous to man " is incorrect [this *Bulletin*, 1947, v. 44, 7–12]. p. 147—writing of the life-cycle of a tick the author states " Nymphs hibernate unfed. The following year they suck blood from rodents or man, drop to the ground, and transform into eight-legged adults. The young adults hibernate unfed. The following year they feed on larger animals or man." This brief account, as applying to the general life-cycle of Ixodid ticks, is obviously inaccurate and misleading. p. 153—three species of *Ornithodorus* are mentioned as vectors of relapsing fever, but no mention is made of *O. moubata*.

The book concludes with a very useful vocabulary of technical terms in which the origins of some 350 technical terms and scientific names are analysed.

There is a good colour plate depicting the malaria parasite, and the majority of the text figures have been borrowed from reliable sources, but in some instances, notably the drawing of *Phlebotomus* (p. 182), these have suffered in reproduction. There is an adequate index and the printing, binding and general presentation of the book are excellent. *R. M. Gordon*

CHANDLER, Asa C. [M.S., Ph.D., Professor of Biology, Rice Institute, Houston, Texas, etc.]. **Introduction to Parasitology with special reference to the Parasites of Man.** Eighth Edition. pp. xii+756, 273 figs. 1949. New York : John Wiley & Sons, Inc. London : Chapman & Hall, Ltd. [48*s*.]

The welcome appearance of an 8th edition of this book reflects the rapid advances of parasitology which have occurred since 1944, when the 7th edition [see this *Bulletin*, 1946, v. 43, 1085] was issued. Its author says in his preface that more advances have been made during this time than in any similar period since the book was first published and there are, therefore, changes on every page of the present edition. There are also new sections on bacteria, rickettsiae and filterable viruses transmitted by arthropods, and on insecticides and repellents. A separate chapter is now given to the Acanthocephala and new keys to some of the arthropod species have been added. Veterinary readers will welcome the more extensive consideration of species parasitic in domesticated animals. Some of the illustrations are new and, as the author claims, most of them have been improved. This improvement enhances the value of the book, but some readers who find it the most readable and entertaining of all the textbooks on parasitology, would like to see the illustrations still better. Some of them might be bigger, and better printing of many of the line drawings would remove the rather harsh quality that they have in the copy under review.

The arrangement of the remarkably comprehensive contents of the book has not been altered. The first two chapters state the general principles of parasitology. A useful feature of the second chapter is its general account of immunity to parasitic animals, an important subject whose recent progress is indicated at appropriate places throughout the book. The succeeding chapters discuss the various groups of parasites, a chapter on spirochaetes being included. The discussion of each of these groups begins with a general account and proceeds to a detailed consideration of the structure of species encountered by the medical man and the veterinarian and of their pathology, symptoms, epidemiology and treatment. At the end of each chapter there is a valuable bibliography and a final section lists the most important journals in which literature on parasitology can be found. The index, always an important feature of a book of this kind, is carefully done, but items such as " Flesh flies, see Sarcophagidae " give the reader unnecessary trouble which could be avoided by printing the reference under both of these headings.

Throughout the book there are valuable references to the physiology of parasites, so that the reader gets, in addition to morphological detail, directives towards the physiological parasitology which is rapidly growing and may in the future revolutionize methods of control and treatment of parasitic diseases. Another feature of the book is the picture that it gives us of the effects of parasites on some aspects of human civilization. The author's graphic and entertaining style and his practical experience of the battle against parasites in various countries enable him to draw this picture effectively and accurately. Now and then, in order, no doubt, to economize words and to give them force, he uses a teleological phraseology which is, some readers may feel, out of place in a scientific book ; but this can do little harm in a work which will hardly be read by unintelligent people. More important is the author's success in correcting the widespread impression that textbooks are certain to be dull. To have done this without any sacrifice of scientific quality is a considerable achievement. *G. Lapage*

GIORDANO, Mario. **Patologia, parassitologia ed igiene dei paesi caldi.** [Pathology, Parasitology and Hygiene of Warm Climates.] 3rd Edition. Vol. I. pp. xxiv+1–812 & Vol. 2. pp. 813–1627, 598 figs. & 8 coloured pls. 1950. Rocca San Casciano : Cappelli. [Lire 12.000 (2 volumes).]

A little more than ten years has passed since the previous edition of this work was issued [see this *Bulletin*, 1940, v. 37, 154] ; momentous years which have included the waging of a world-wide war in which much of the fighting took place in tropical and subtropical countries by troops trained in temperate climates and exposed for the first time to tropical conditions, hazards of climate and risks of infection unknown in the countries whence they came.

Experience gained by medical men working with such troops and amid unwonted surroundings has been recorded in hundreds of articles, papers, reports and communications, all of which had to be considered and those deemed to be sufficiently important had to be embodied in any work pretending to be authoritative and up to date.

It is not surprising, therefore, that this work by Professor Giordano has increased by more than 50 per cent., from a little over 1,000 pages to more than 1,600 ; that many sections have had to be revised and in part rewritten, interpolations made and sections added. The whole type has been changed, each page contains more lines, the type headings of sections are altered for the better, sections raised to the status of separate chapters—all tending to ease of reading, and two, more handy, volumes take the place of one bulky tome.

The general arrangement of the work has been retained, but, whereas the second edition consisted of ten parts with an introductory section and an appendix on Sanitary Laws and Regulations, the third comprises twelve parts, the introductory section becoming Part I and the Laws and Regulations, Part XII.

Part I now comprises three chapters in place of two in the second edition. The first, on Climatology, contains additions defining the different types of climate and the action and effect of warm climates on the human functions and systems, and this chapter has increased in size. Chapter 2 is a useful addition, on the selection of those who propose to settle in warm countries and points are given for the guidance of medical men who are called upon to make the decision. Chapter 3, on general hygienic measures, the best season for going out to the tropics, hints on clothing, dwellings, food, water-supply, hours of work, etc., has been nearly trebled in size, from 7 to 20 pages.

The protozoological chapters of Part II have increased this section of the work by more than half, from 302 pages to 478. Each subsection has evidently

been subjected to careful thought and revision, with many additions, too numerous for detailed mention. The modes of reproduction of Protozoa are now inserted. The treatment of relapsing fever, formerly restricted to a few remarks on arsenicals, has been amplified to include the use of penicillin, chloromycetin, serum therapy and the treatment of complications. The leptospirosis section now includes an account of the various leptospires found in Europe and an addition on "arthritic spirochaetosis," Reiter's disease. A description of the pathological anatomy of Castellani's broncho-pulmonary spirochaetosis and an illustration of a lung with cavitation due to the association of the spirochaete with *F. fusiformis* are now included.

In several places throughout this work—Yaws is an example—the order of the illustrations has been changed and comparison with the former edition will show that many of the illustrations have been printed left to right or upside down.

Goundou was formerly mentioned incidentally under yaws but was given a separate chapter in the part dealing with diseases of undetermined virus origin; now it follows yaws and the author states: "If we exclude yaws, the cause remains unknown". Other spirochaetoses have a somewhat different order from that of the last edition and each section has been revised, but not much changed otherwise. In the Rhizopod section, the account of the symptomatology of amoebic dysentery has been enlarged by inclusion of Castellani's three characteristic signs: subensifrom pain, the mid-axillary sign, and the limited zone of dullness with increased vocal fremitus at the base of the right lung. In the Leishmania section, the American visceral form due to *Leishmania chagasi*, thought to be a distinct species, was referred to in a few lines in the previous edition, but now has a whole page devoted to it. The use of new drugs has been detailed in the African trypanosomiasis chapter.

The malaria sections are much improved. The work of SHORTT and GARNHAM on the pre-erythrocytic stages of the parasite has been embodied; there is a new map of the geographical distribution of malaria, an account of *Plasmodium ovale* has been added and many of the new drugs tried in the treatment of malaria during the last ten years find a place; the same applies to the use of insecticides in control. In the chapters dealing with rickettsioses there are fresh additions on diseases allied to Rocky Mountain spotted fever and one on Q fever, which, absent from the second edition, is fairly fully dealt with in this. Trachoma has been removed from Parasitic Diseases of Doubtful Origin to virus diseases along with yellow fever, rabies, dengue, variola, epidemic hepatitis and psittacosis.

Part III, Helminthology, is one of the best parts of the book, but the ovum of *Ancylostoma duodenale* is still depicted with a double wall, and *Taenia solium*, which is rightly said to have a double row of hooklets on the scolex, is depicted with a single row and the characteristic shape of the hooklets is not mentioned either of this or any other species in which the hooklets are diagnostic.

Entomology, Part IV (formerly Part III) is little changed, except for rearrangement of some of the figures, omission of one or two which showed little, and there is a new coloured plate of *Triatoma megista*. Other changes consist mainly of amplifications in the text, too numerous to specify. One important addition is that the treatment of *Sarcoptes scabiei* infection has been much enlarged to include half a dozen methods or preparations which have been tried since the previous edition was published. The Myriapoda instead of being relegated to the position of a mere subsection have now a separate chapter.

As in the second edition, the sections on Poisonous Fish and Vegetal Poisons remain the weakest of the book. One ought not, perhaps, to expect one man to be master of the many branches of tropical medicine, but to dismiss all the poisonous fish in 2½ pages is almost a travesty. In fact, the section consists of

little more than general remarks and the only one depicted is *Thalassophrina maculosa* and that very obscurely. Vegetal poisons occupy 18 pages altogether ; the whole section is sketchy and incomplete ; it will be noticed that there are important omissions and the information when it is given is scanty.

Part VI, Diseases due to Bacteria, is as full as could be expected in a general work on tropical diseases. In an introductory section are brief remarks on pneumonitis, cerebrospinal fever, erysipelas and tuberculosis and there is a new subsection on bacillary enteritis, botulism and diphtheria. Castellani's tropicaloid ulcer receives a full description—aetiology, diagnosis and treatment. The treatment of leprosy has been brought up to date, but ainhum is still included under leprosy (though earlier it is remarked *en passant* " some authors think it is related to yaws ").

In Part VII, devoted to Deficiency Diseases, the vitamin section has been amplified to include new discoveries ; also sprue, formerly among the mycoses, now finds its proper place. Pinta also has been transferred from the Mycosis section, as has also Gangosa. A chapter has been added on War Oedema. The Mycosis section is good and has been brought up to date and considerably re-arranged. It will be observed that dhobie mark dermatitis is mentioned and its possible confusion with dhobie itch ; the former a dermatitis venenata due to juice of the Bela gutti nut, the latter due to *Tinea cruris*.

In yellow fever the entomological advances of the past decade are mentioned and prophylactic vaccination, formerly disposed of in a page and a half, now occupies three times this and there is a record of its preparation, use and results. A separate chapter is given up to other virus diseases which may be met with—measles, scarlet fever, german measles, mumps, influenza, poliomyelitis, forms of encephalitis and encephalo-myelitis, infectious mononucleosis and others.

Lastly, new Laws and Regulations have necessitated the final part being increased from 100 to 160 pages. There are many other good points in this work which lack of space precludes from mention and we can only sum up by saying that this continues to be one of the best one-man books on the diseases of warm climates. *H. Harold Scott*

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Am.	signifies	Amoebiasis and Intestinal Protozoal Infections.	Lep.	signifies	Leprosy.
Bart.	,,	Bartonellosis.	Mal.	,,	Malaria.
Bl.	,,	Blackwater Fever.	Misc. Dis.	,,	Miscellaneous Diseases.
B.R.	,,	Book Review.	Oph.	,,	Tropical Ophthalmology.
Chl.	,,	Cholera.	Pl.	,,	Plague.
Def. Dis.	,,	Deficiency Diseases.	Prot.	,,	General Protozoology.
Den.	,,	Dengue and Allied Fevers.	Rab.	,,	Rabies.
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Hel.	,,	Helminthiasis.	Ulc.	,,	Tropical Ulcer.
Lab.	,,	Laboratory Procedures.	Vms.	,,	Venoms and Antivenenes.
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Benhamou, E., Albou, A., Destaing, F. & Puglièse, J., 830 (Leish.)
Beninson, J., with Anigstein & Whitney, (29) (Typh.)
Benitez Calvo, L. A., 482 (Hel.)
Benitez-Gautier, C., with Suarez, Spies, Hernandez-Morales & Perez, 397 (Sp.)
Bennett, B. L., with Crocker, Jackson, Snyder, Smadel, Gauld & Gordon, 617 (Typh.)
——, Smadel, J. E. & Gauld, R. L., 223 (Typh.)
Bennett, H. J., with Hunter, Fry, See & Greene, 380 (Hel.)
Bennison, B. E. & Coatney, G. R., 524 (Mal.)
Bentley, C. A., 4 (Mal.)
Béquignon, R. & Vialat, C., 352 (Rab.)
Berberian, D. A. & Dennis, E. W., 114 (Mal.)
——, with —— & Hansen, 243 (Am.)
Berge, T. O., Gauld, R. L. & Kitaoka, M., 349 (Typh.)
——, with Kuwata & Philip, 531 (Typh.)
Bergeret, C., 9 (Mal.), 48 (R.F.), 265 (Def. Dis.)
—— & Raoult, A., 46 (R.F.)
Bern, H. A. & Hansen, M. F., 1052 (Mal.)
Bernal Flandes, F., 155 (Vms.)
Bernard, F., with Mandoul & Jacquemin, 896 (Ent.)
Bernard, L., with Cavaillon, Adam, Camain, Jaujou & Trinquier, 1179 (Mal.)
Bernardino, C. E., with Dy & Bernardo, 1178 (Mal.)
Bernardo, B. C., with Dy & Bernardino, 1178 (Mal.)
Bernet, 596 *bis* (Mal.)
Bernet, J., 195 (Mal.)
Berry, E. G., with Nolan, 143 (Hel.)
Berti, A. L., with Stage, (815) (Mal.)
Berti, F. A., with Rheinboldt, Perego, Rieckmann, Rzeppa, Mauri, Hadler & de Souza Lima, 548 (Lep.)
——, Rieckmann, B., Perego, C. & Rzeppa, H. W., 372 (Lep.)
Bertram, D. M., 814 (Mal.)

Bertram, D. S., 482, 874 (Hel.)
Bertrand-Fontaine, Fauvert, R., Schneider, J. & Hartmann, L., 433 (Mal.)
——, Schneider, J. & Rivron, J., 532 (Typh.)
——, ——, Wolfromm, R. & Cagnard, V., 67 (Hel.)
Bettencourt-Gomes, S. C., 398 (Haem.)
Beyra Alemany, M., with Ortiz Machado, (1057) (Mal.)
Bezian, A., with Pautrizel & Bailanger, 375 (Hel.)
Bhandari, R. M., 649 (Hel.)
Bhansali, K. M. & Kothari, G. J., 614 (Typh.)
Bhatia, M. L., 565 (Ent.)
Bhatt, H. R., 952 (Mal.)
Bhatt, P. K., (158) (Misc. Dis.)
Bhattacharjea, S. K., (1019) (Hel.)
Bhende, Y. M., Purandare, N. M., Banker, D. D., Figueredo, N. & Desai, S. D., 337 (Leish.)
Bianco, I., 711 (Mal.)
Bibile, S. W., Cullumbine, H., Kirtisinghe, C., Watson, R. S. & Wikramanayake, T., (87) (Misc. Dis.)
——, ——, ——, Wickramanayake, T. W. & Watson, R. S., (77) (Haem.)
——, ——, Watson, R. S. & Wikramanayake, T. W., 168 (Reports, etc.)
——, with —— & Wikramanayake, (1216) (Def. Dis.)
Bieling, R., 457 (Typh.)
—— & Oelrichs, L., 344, 345 *bis* (Typh.)
Billington, V. L., 521 (Mal.)
Binaghi, G., 755 (Lep.)
Bincer, W., 593 (Mal.)
Binder, T., with Sicé, 805 (Mal.)
Biocca, E., 802 (Mal.)
Birkett, B., with Bishop, 524 (Mal.)
Birsner, J. W., with Miller, 401 (Der.)
——, with Smale, 85 (Der.)
Birtwell, S., (206) (Mal.)
——, Curd, F. H. S. & Rose, F. L., (205) (Mal.)
Bishop, A. & Birkett, B., 524 (Mal.)
—— & McConnachie, E. W., 525 *ter*. (Mal.)
Bishopp, F. C., (782) (Ent.)
Bittner, G., 570 (Lab.)
Bivens, M. D., 1133 (Reports, etc.)
Blaauw-van Dok, S., with Stoppelman, 781 (Prot.)
Blaché, R., with Montestruc & Caubet, 1094 (Lep.)
——, with —— & Laborde, 1113 (Hel.)
Black, J., with Koprowski, 846 (Rab.)
Black, R. H., 1217 (Def. Dis.)
Blackie, W. K., 1143 (B.R.)
Blackith, R. E., with Page, 93 (Ent.)
——, with —— & Stringer, 93 (Ent.)
Blagg, W., with Dawson, 739 (Chl.)
Blanc, F., 113 (Mal.)
—— & Languillon, J., 6 (Mal.)
—— & Touzin, R., 1104 (Hel.)
Blanc, G., Bruneau, J. & Chabaud, A., 564 (Prot.)
——, —— & Pagès. R., 663 (Oph.)
——, Martin, L. A. & Bruneau, J., 130 (Typh.)
Blanco Rabassa, E., with Basnuevo, 57 (Hel.)
Blankenhorn, M. A., 265 (Def. Dis.)
Blood, B. D., 734 (Rab.)
Bloomfield, R. A., with Mercer, Lund & Caldwell, 1109 (Hel.)
Bloss, J. F. E., 146 (Hel.)
Blum, L., with Most, Kane, Lavietes, Schroede Behm, Katzin & Hayman, 1009 (Hel.)
Blumberg, N., Doane, J. C. & Weiss, L. B., (2 (Typh.)
Blumenthal, C. J., 1127 (Oph.)
Boari, D., 1209 (Hel.)
Boccalandro, C. A. & Esquivel, R. B., 352 (Rab
Boccia, G., (127) (Typh.)
Bock, M., 362 (R.F.)
——, with Kikuth, 227 (Typh.)
Bocobo, F. C., 156 (Der.)
Bodenlos, L. J., with Perry, (786) (Ent.)
Boecker, E., 131 (Rab.)
Boehncke, H., 875 (Hel.)
Boenjamin, R., 363, 472, (552) (Lep.)
——, with Lampe, 543 (Lep.)
Bogliolo, L., 660 (Der.)
Bohart, R. M. & Ingram, R. L., 1040 (Ent.)
Boiron, H., 362 (R.F.)
Boldt, T. H. & Goodwine, C. H., 320 (Mal.)
Bol. Oficina Sanitaria Panamericana, (733) (73 (Rab.)
Bonar, S. C., with Obermayer & Rosenqui 1098 (Lep.)
Bonis, M., 225 (Typh.)
Bonnel, D., with Lamy, 165 (Lab.)
Bonnet, D. D., with Gross, 461 (Pl.)
Bonnet, P., (1032) (Misc. Dis.)
Bonnin, H., 989 (Am.)
—— & Moretti, G. F., 1108, 1115 (Hel.)
——, —— & Guignard, J., 742 *ter*. (Am.)
Booker, C. G., 172 (B.R.)
Bookless, A. S., 626 (Am.)
Boquet, P., Dworetzky, M. & Essex, H. E (1030) (Vms.)
Bordas, E., with Downs, 429, 587 (Mal.)
Bordes, L. A., (467) (Am.)
Borgeix, L., with Clerc, 136 (Am.)
Borgers, G., with Wanson & Pannier, 873 (He
Borges, G. B., 587 (Mal.)
v. Bormann, F., 833, 1189 (Typh.)
Borrotchin, M., with Pellegrino, 23 (Tryp.)
Borsoni, G., with Dana, Sebag & Cohen, 10 (Typh.)
Bose, A. N., Bose, S. & Ghosh, T. N., 11 (Am.)
Bose, J. P., with Chakravarty, Sen Gupta & D 726 (Leish.)
Bose, S., with Bose & Ghosh, 1198 (Am.)
Boshell-Manrique, J., with Cerqueira, 130 (Y.F
Botreau-Roussel, (998) (Ys.)
——, Ribo & Sua, H. B., (998) (Ys.)
Boulard, G., with Thiodet, Fourrier & Raffi, 1 (Mal.)
Bouquet, A., with Kerharo, 1102 (Lep.)
Bourgain, M., 499 *bis* (Ent.)
Bourke, A. R., with Robbins & Smith, 9 (Typh.)
Bourne, G. H., 772 (Def. Dis.)
Bouvier, G., 1131 (Ent.)
Bovarnick, M. R. & Miller, J. C., (970) (Typh.)
——, —— & Snyder, J. C., 832 (Typh.)
Boventer, K., 251 (Hel.)
Bowie, J. H., with Nutt, Wright & Beattie, (Prot.)
Box, L., with Hoekenga, 390 (Hel.)
Boyce, S., with DeMarsh, 1218 (Haem.)
Boyd, G. A., with Watts, 1105 (Hel.)

Boyd, J. S. K., 416 (Reports, etc.)
Boyd, M. F., 502 (B.R.)
Boyer, J., (728) (Typh.)
Bozicevich, J., 981 *bis* (Am.)
Bradin, J. L., Jr. & Hansen, E. L., 538 (Am.)
Bradley, G. H. & Goodwin, M. H., Jr., 301 (Mal.)
Brady, F. J., with Ashburn & Burch, 389 (Hel.)
——, with Luttermoser & Haskins, 254 (Hel.)
von Brand, T., 1033 (Prot.)
——, Mehlman, B. & Nolan, M. O., 56 (Hel.)
——, Tobie, E. J., Kissling, R. E. & Adams, G., 218 (Tryp.)
——, with —— & Mehlman, 604 (Tryp.)
Brandão, C. H., with Arantes, (78) *bis* (Vms.)
Bras, G., with Lie Kian Joe, 861 (Hel.)
——, Rijkebüsch, L., Kotter, G. F. & Djoa Liang Ham, 83 (Der.)
Brauneck, H. W. F., 274 (Misc. Dis.)
Brawley, R. W. & Modern, F. W. S., 348 (Typh.)
Brazzaville, 718 (Tryp.), 899 (B.R.)
Brede, H. D., 392 (Hel.)
Breslin, D., with Rigdon, 602 (Mal.)
Breuer, H., (Hel.)
Brewer, A. E., with Conan & Head, 992 (Am.)
Briceño Rossi, A. L., 232 (Rab.), 769 (Hel.)
—— & Hewitt, R., 387 (Hel.)
—— & Mayer, M., 644 (Hel.)
Brink, A. J., (86) (Misc. Dis.)
Brinkman, G. L. & Prior, I.A.M., 1121 (Def. Dis.)
Briscoe, M. S., 305, (1054) (Mal.)
British Med. J., 75 (Sp.), 673 (Reports, etc.)
British Museum (Natural History) (497) *bis*, (499) (Ent.)
Brito, R. S. & Cotrim, J., 1174 (Mal.)
Brodine, C. E., with Becker & Marousek, 325, 603 (Mal.)
Brooke, M. M., 981 *ter* (Am.)
—— & Donaldson, A. W., 518 (Mal.)
Brooks A. G., d'Silva, C. B., Lahiri, B. N. & Ahuja, M. L., (842) (Rab.)
Broscheit, A., with Hötz, 1018 (Hel.)
Brown, E., with Hauschka, Goodwin & Palmquist, 607 (Tryp.)
Brown, H. W., 981 (Am.)
——, with Hussey, 981 (Am.)
Brown, J. H. & Kohls, G. M., (897) (Ent.)
Brown, M., Cronk, L. B., deSinner, F., Green, J. E., Gibbons, J. E. & Kuitunen-Ekbaum, E., 558 (Hel.)
Brownlee, G., Goss, M. D., Goodwin, L.G., Woodbine, M. & Walls, L. P., 1070 (Tryp.)
Bruce, H. M. & Parkes, A. S., (165) (Lab.)
Bruce-Chwatt, J. M., with Bruce-Chwatt, 956 (Mal.)
Bruce-Chwatt, L. J., 951 (Mal.)
—— & Bruce-Chwatt, J. M., 956 (Mal.)
—— & Kee Hock Ooi, 1042 (Ent.)
Brummer, P., 540 (Am.)
Brumpt., E., 573 (B.R.)
Brumpt, L. C., (1060) (Mal.)
—— & Chabaud, A. G., 148 (Hel.)
——, with Galliard, 58 (Hel.)
——, with —— & Martinez, 888 (Misc. Dis.)
Bruneau, J., with Blanc & Chabaud, 564 (Prot.)
——, with —— & Martin, 130 (Typh.)
——, with —— & Pagès, 663 (Oph.)
—— & Nguyen-Dinh-Diep, (975) (Tpyh.)
Brygoo, E., 757 (Lep.)
——, with Harant, (90) (Ent.)
Bueding, E., (140) (Hel.), 1035 (Prot.)
—— & Koletsky, S., 863 (Hel.)
——, with Ross, 633 (Hel.)
Buisson, B., with Schneider, Marchalant, Lotte & Diallo, 449 (Tryp.)
Bülbring, E., Lourie, E. M. & Pardoe, U., 119 (Tryp.)
Bull. Méd. de l'Afrique Occidentale Française. (Journées Méd. de Bobo-Dioulasso 24 au 29 Jan. 1949), 449 (Tryp.)
Buonomini, G. & Maccarrone, A., 439 (Mal.)
Burch, T. A., with Ashburn & Brady, 389 (Hel.)
Burchenal, J. H., with Southam & Thomson, 392 (Hel.)
Burgess, R. W., with Coatney, Cooper, Ruhe & Young, 1171 (Mal.)
Burgio, G. R., with d'Alessandro & Mariani, 831 (Leish.)
Burke-Gaffney, H. J. O'D., 169 (B. R.)
Burlingame, P. L., 866, 867 (Hel.)
—— & Gardner, H. T., with Resemann, G., Cranmer, J. & Vollmer, E., 358 (Am.)
Burnip, R., with Cohen, 493 (Der.)
Burrows, W., with Koshland, 1083 (Chl.)
Burtt, E., 606 (Tryp.)
de Bustamante, F. M., Aguirre, G. H. & Filho, J. C. A., 815 (Mal.)
——, Costa, J. L. & Azambuja, C. E. A., 587 (Mal.)
—— & Pires, W. da M., 897 (Ent.)
——, with Rachou & Ferreira, 600 (Mal.)
Busvine, J. R., 599 (Mal.)
—— & Tew, R. P., 894 (Ent.)
Butts, D. C. A., 1057 (Mal.)
Butz, W. C. & Elton, N. W., (1226) (Misc. Dis.)
Buxton, P. A., 1214 (Hel.)
Bylmer, H. J. T., 202 (Mal.)

C

Cabassu, H., with Ranque & Ranque, 341 (Leish.)
Cabrera, D. J., with Smith & Dy, 1173, 1174 (Mal.)
Cagnard, V., with Bertrand-Fontaine, Schneider & Wolfromm, 67 (Hel.)
Caldeira, R. da G., 51 (Lep.)
Caldwell, F. E., with Mercer, Lund & Bloomfield, 1109 (Hel.)
Calero C., 26 (Typh.)
Calero M., C. & Ortiz O., P., 651 (Hel.)
Cali', G., with Scaffidi, 128 (Typh.)
Callot, J., Vermeil, C. & Puech, J., 618
Calverley, E. T., 577 (B.R.)
Calvó Fonseca, R., 620 (Rab.)
Camain, R., (284) (Reports, etc.)
——, with Cavaillon, Adam, Bernard, Jaujou & Trinquier, 1179 (Mal.)
Cambournac, F. J. C., 1180 (Mal.)
Cameron, I. G., 675 (Reports, etc.), 730 (Typh.)
—— & van Someren, G. R. C., 764 (Hel.)
Cameron, T. W. M., 273 (Misc. Dis.), (675) *bis* (Reports, etc.)
Cami, R., with Sautet & Asseo, 36 (Am.)
Camin, J. H., (566) (Ent.)
Caminopetros, J., 534 (Typh.)
Campbell, A. G., with Humphreys, Driver & Hatton, 751 (R.F.)

Campbell, N. R., with Collier & Fitzgerald, 980 (Chl.)
Camphyn, R., with Wanson, 537 (Pl.)
Campillo, C., 620 (Rab.)
Campins, H., Scharyj, M. & Gluck, V., 775 (Der.)
Campo Posada, A., 386 (Hel.)
Campos R. de C., J., 247 (Lep.)
Camus Gundián, D., 998 (Lep.)
Cancellara, E., 883 (Oph.)
Canet, J., 205, 206 (Mal.), 242 (Am.)
—— & Jahan, P., 261 (Hel.)
Cantrell, W. & Jordan, H. B., 118 (Mal.)
Capponi, M., with Gallais & Cros, 7 (Mal.)
Cardenas Montero, A., (1109) (Hel.)
Cardoso, F. A. & de Carvalho, P. E., 33 (Hel.)
Carne, H. R., with Wickham, 668 (Prot.)
Caron, M., with Harant, 391 (Hel.)
Carpenter, C. M., Stokinger, H. E., Suhrland, L. G. & Ackerman, H., 52 (Lep.)
Carpenter, E., with Lincicome & Thiede, 987 (Am.)
Carrasco, E. O., with Salcedo, Bamba, Jose & Valenzuela, 560 (Def. Dis.)
Carrera, G. M., 988 (Am.)
—— & Faust, E. C., 465 (Am.)
——, with Sadun, Krupp & Allain, 852 (Am.)
Carrijo, L. N., with Travassos & Rodrigues, 128 (Typh.)
Carson, M. J., Gowen, L. F. & Cochrane, F. R., 225 (Typh.)
Carter, M. G. & Korones, S. B., (626) (Am.)
de Carvalho, F., with de Azevedo & Coelho, 1003 (Hel.)
de Carvalho, J. D., 480 (Hel.)
de Carvalho, P. E., with Cardoso, 53 (Hel.)
Casanges, A. H., McGovran, E. R. & Chiles, J. V., (432) (Mal.)
Casile, M., (138) (Lep.)
Castellani, A., 1032 (Misc. Dis.)
Castriota, L., with Andrini, 768 (Hel.)
Casula, D., 1171 (Mal.)
Catanei, A., with Parrot, 428, 1051, 1168 (Mal.)
Cates, E. C., (735) (Rab.)
Cathie, I. A. B. & Dudgeon, J. A., 496 (Prot.)
Cattan, R., (539) (Am.)
Caubet, P., with Montestruc & Blaché, 1094 (Lep.)
Causey, O. R., with Damasceno & Arouck, 1231 (Ent.)
——, Kumm, H. W. & Laemmert, H. W., Jr., 977 (Y.F.)
——, with Laemmert & Hughes, 30 (Y.F.)
Cavaillon, A., Adam, J. P., Bernard, L., Camain, R., Jaujou, C. & Trinquier, E., 1179 (Mal.)
Ceccaldi, J., 718 (Tryp.)
—— & Arnoult, H., 19 (Tryp.)
——, —— & Pellissier, A., 528 (Tryp.)
——, Pellissier, A. & Arnoult, H., 18 (Tryp.)
——, Trinquier, E. & Arnoult, H., 822 (Tryp.)
——, ——, —— & Menu, P., 824 (Tryp.)
——, ——, Pellissier, A. & Arnoult, H., 528 (Tryp.)
Cerqueira, N. L. & Boshell-Manrique, J., 130 (Y.F.)
Céspedes F., R., with Pizzi, 467 (Am.)
Ceylon, 1136 (Reports, etc.)
Chabaud, A., with Blanc & Bruneau, 564 (Prot.)
Chabaud, A. G., 670 (Ent.)
——, with Brumpt, 148 (Hel.)
Chabaud, H. E. & Morin, H. G. S., (975) (Typh.)
Chaigneau, H., with Levaditi, Vaisman & Bar 1103 (Lep.)
Chakrabarty, A., (45) (R.F.)
Chakravarty, N. K., with Chaudhuri, 958 (M 1029 (Ep. Dropsy.)
——, Sen Gupta, P. C., Bose, J. P. & De, U. 726 (Leish.)
Chambers, W. L., with Legerton, 470 (R.F.)
Champeau, M. F., 657 (Der.)
Chan, A., with Hewitt, Kenney & Mohamed, (Hel.)
Chanarin, I., 275 (Misc. Dis.)
Chandler, A. C., 1237 (B.R.)
Chandler, R. L., with Fulton & Joyner, 1 (Leish.)
Chang, E., 664 (Oph.)
Chang, L. W., 661 (Oph.)
Chang, T. L., with Hsu & Feng, 952 (Mal.)
Chardome, M. & Peel, E., 65 (Hel.)
——, with van den Berghe, 644 (Hel.)
——, with —— & Vincke, 816 (Mal.)
Charlewood, G. P., Shippel, S. & Renton, 54 (Hel.)
Charters, A. D., 540 (R.F.), 745 (Am.)
Chassain, J., with Kernevez, 825 (Tryp.)
Chatterjee, A. K., (269) (Vms.)
Chatterjee, P. K. & Sen Gupta, S., (135) (Am.
Chaudhuri, R. N. & Chakravarty, N. K., (Mal.), 1029 (Ep. Dropsy)
—— & Rai Chaudhuri, M. N., 1175, 1176 (M
Chaussinand, R., 362, (998) *bis*, (1102) (Lep.)
——, with André-Thomas & de Ajuriaguerra, (Lep.)
Chelarescu, M., with Ciuca, Ballif, Timise Vasiliu-Munteanu & Trofim, M. V., 593 (M
Chellappah, L. R., with Reid & Rajamoney, (Mal.)
Chemnitz, H. & Kirsch, E., 337 (Leish.)
Chen, C. Y., with Tokura, Tsai & Lo, 604 (M
Chen, G., with Reilly & Geiling, 326, 717 (M
Chen, H. T., 1103 (Hel.)
Chen, R. T. S., 356 (Pl.)
Chen, T. T., 340 (Leish.)
Cheng, W. J., with Ling & Chung, 380 (Hel.)
Cheriyan, O., with Balasubrahmanyan, 467 (A
Chesterman, C. C., 1090 (Am.), 1214 (Hel.)
Chetty, K. N., with Subramaniam, (950) (Mal
Ch'i, C. & Zia, S. H., 738 (Chl.)
Chian, T., with Wu & Wan, 665 (Ulc.)
Chiles, J. V., with Casanges & McGovran, (4 (Mal.)
Chini, V. & Valeri, C. M., (77) (Haem.)
Chorley, J. K., 966 (Tryp.)
Chotkowski, L. A., 154 (Vms.)
Chow, C. Y., (9), 432, 518, 586 (Mal.), 1040 (E
—— & Balfour, M. C., 709 (Mal.)
Chowdhuri, B. N. & Mukerji, B., (50) (Lep.)
Christen, R., with Jarpa, Agosin & Pizzi, (Tryp.)
——, with Pizzi, Agosin, Hoecker & Neghme, (Tryp.)
Chronicle World Health Organization, 622 (Pl
Chu, I. T., with Wu & Wu, 339 (Leish.)
Chung, H. L., with Ling & Cheng, 380 (Hel.)
Chung-Hoon, E. K., with Sloan, Godfrey-Ho & Hedgcock, 1098 (Lep.)
Ciaccio, G., with Giroud, 613 (Typh.)
——, with —— & Pan, 613 (Typh.)
——, with —— & Vargues, 613 (Typh.)

Cifarelli, F., with Amalfitano, Benetazzo, Gambini, Servino & Tarizzo, 123 (Leish.)
Cilesiz, A., 234 (Rab.)
Ciuca, M., Ballif, L. & Chelarescu, M., with Timisescu, A., Vasiliu-Munteanu, F. & Trofim, M. V., 593 (Mal.)
Clark, J. C., with Smith, Cole & Jones, 279 (Ent.)
Clarke, J. L., 893 (Ent.)
Clavel, B., with Ravina, Guérin & Pécher, (989) (Am.)
Clavero, G., with Perez Gallardo & Hernández Fernández, 840 (Typh.)
—— & Romeo Viamonte, J. M., (803), 812 (Mal.)
Clavero Del Campo, G. & Perez Gallardo, F., 222 (Typh.)
Clerc, S. & Borgeix, L., 136 (Am.)
Clier, J. L., with Gaud & Méchali, 595 (Mal.)
Clow, J. M., with Smith, 135 (Am.)
Coatney, G. R., with Bennison, 524 (Mal.)
——, with Cooper, Culwell, Eyles & Young, 1172 (Mal.)
——, Cooper, W. C., Culwell, W. B., White, W. C. & Imboden, C. A., Jr., 1177 (Mal.)
——, with —— & Imboden, 1057 (Mal.)
——, with ——, —— & Jeffery, 438 (Mal.)
——, with —— & Ruhe, 314 (Mal.)
——, ——, Ruhe, D. S. & Young, M. D., 318 (Mal.)
——, ——, ——, —— & Burgess, R. W., 1171 (Mal.)
——, ——, White, W. C., Lints, H. A., Culwell, W. B. & Eyles, D., 1060 (Mal.)
——, Greenberg, J., Cooper, W. C. & Trembley, H. L., 444 (Mal.)
——, with —— & Trembley, 603 (Mal.)
——, with Lastra, 1068 (Mal.)
——, with Lints, Cooper, Culwell, White & Eyles, 1059 (Mal.)
——, with Spicknall & Terry, 318 (Mal.)
Coblentz, B., with Siguier, Piette, Lemenager & Trélat, 1173 (Mal.)
Cochrane, F. R., with Carson & Gowen, 225 (Typh.)
Coda, D., 207 (Mal.)
—— & Ramos, A. da S., 430 (Mal.)
Coelho, M. F., with de Azevedo & de Carvalho, 1003 (Hel.)
Coggeshall, L. T., with Yount, 435 (Mal.)
Coghill, N. F., 45 (R.F.)
Cohen, J., with Dana, Sebag & Borsoni, 1074 (Typh.)
Cohen, R. & Burnip, R., 493 (Der.)
—— & Miller, L., 1031 (Der.)
Cohen, S., with Murray, Baehr, Shwartzman, Mandelbaum, Rosenthal, Doane, Weiss & Snyder, 729 (Typh.)
Colaço, A. T. F., 1005 (Hel.)
Colas-Belcour, J., with Roubaud & Rageau, 606 (Tryp.)
—— & Vervent, G., 361, 362 (R.F.)
Colbert, J. W., Jr., Strauss, M. J. & Green, R. H., 778 (Der.)
Colbourne, M. J., (768) (Hel.)
Cole, C. R., with Prior & Torbet, 399 (Der.)
Cole, M. M. & Smith, C. N., 783 (Ent.)
——, with ——, Jones & Clark, 279 (Ent.)
Coleman, S. A., with Penn (306) (Mal.)
Coll, H. A., with Alvarado & Laguzzi, 716 (Mal.)
Collari, S., 594 (Mal.)
Colless, D. H., 109 (Mal.)
Collier, H. O. J., Campbell, N. R. & Fitzgerald, M. E. H., 980 (Chl.)
——, Hall, I. F. & Waterhouse, P. D., 236, 1196 (Chl.)
—— & Waterhouse, P. D., 1196 (Chl.)
Colonial Office, 180 (B.R.), 279 (Reports, etc.), 1222 (Misc. Dis.)
Colonnello, F., 134 (Am.)
Colorado Iris, R. & Leyva, R. D., 164 (Ent.)
Coluzzi, A., with Raffaele, 105 (Mal.)
Conan, N., Jr., with Most, Miller & Grossman, 1089 (Am.)
Conan, N. J., Jr., 40 (Am.)
——, Head, J. A. & Brewer, A. E., 992 (Am.)
Conant, V., (715) (Mal.)
Conejos, M., with Romaña, Najera & Abalos, 125 (Leish.)
Contreras, F., 372 (Lep.)
——, Guillén, J. & Torrella, E., 369, 373 (Lep.)
Convit, J., with Mayer & Pifano, 611 (Leish.)
——, with Vegas & Espin, 631 (Lep.)
Cook Islands, 166 (Reports, etc.)
Cooney, M., with Adams, Kabler & Adams, 1038 (Prot.)
Coonoor, 572 (Reports, etc.)
Cooper, W. C., Coatney, G. R., Culwell, W. B., Eyles, D. E. & Young, M. D. 1172 (Mal.)
——, with ——, ——, White & Imboden, 1177 (Mal.)
——, with ——, Greenberg & Trembley, 444 (Mal.)
——, —— & Imboden, C. A., Jr., 1057 (Mal.)
——, ——, —— & Jeffery, G. M., 438 (Mal.)
——, with ——, Ruhe & Young, 318 (Mal.)
——, with ——, ——, —— & Burgess, 1171 (Mal.)
——, with ——, White, Lints, Culwell & Eyles, 1060 (Mal.)
——, with Lints, Coatney, Culwell, White & Eyles, 1059 (Mal.)
——, Ruhe, D. S. & Coatney, G. R., 314 (Mal.)
Copp, F. C. & Walls, L. P., (456) (Tryp.)
Copper, A. C., with Verlinde, Makstenieks & Beekman, 1228 (Prot.)
Corcos, A., 988 (Am.)
Cordero Soroa, A., 859 (Lep.)
Corkill, N. L., 393, 1027 (Def. Dis.), 610 (Leish.)
Coronel, L., with Meneghello & Espinoza, 72 (Def. Dis.)
Corradetti, A., 117 (Mal.)
Corrigan, F. L., 59 (Hel.)
Cort, A., with Gear & Wolstenholme, 977 (Typh.)
Cortés, G., with Anselmo, Bahamondes, Ipinza & Lagos, 1216 (Hel.)
Cossery, G. N. & Doorenbos, W., (739) (Chl.)
——, with ——, (739) (Chl.)
Costa, J. L., with de Bustamante & Azambuja, 587 (Mal.)
da Costa, O. R., with Deane, 769 (Hel.)
da Costa, P. D., with da Silva, 376 (Hel.)
Cotrim, J., with Brito, 1174 (Mal.)
——, with Deane, Lédo, Freire, Sutter & de Andrade, 1065 (Mal.)
Courtney, K. O., 1078 (Y. F.)
Courtois, G., 636 (Hel.)
—— & Wanson, M., 634 (Hel.)
Courtois, L., with Wanson & Lebied, 647 (Hel.)

Coutinho, J. O., 710 (Mal.)
Covaleda Ortega, J. & Gállego Berenguer, J., (965) (Mal.)
Covell, G., 11, 707 (Mal.)
——, with Shortt, Fairley, Shute & Garnham, 193 (Mal.)
Covey, J. A., McMahon, J. J. & Myers, H. L., 71 (Hel.)
Cox, H. R., with Koprowski, Miller & Florio, 1080 (Den.)
——, with LeBell, DeBoer & Hazz, 845 (Rab.)
——, with Wong, 29 (Typh.)
Cram, E. B., with Files, 376 (Hel.)
Cranmer, J., with Burlingame, Gardner, Resemann & Vollmer, 358 (Am.)
Crawford, H. E. & Hamman, G. C., 661 (Oph.)
Crewe, W. & Gordon, R.M., 489 (Vms.)
——, with ——, Kershaw & Oldroyd, 1214 (Hel.)
Crocker, T. T., Bennett, B. L., Jackson, E. B., Snyder, M. J., Smadel, J. E., Gauld, R. L. & Gordon, M. K., 617 (Typh.)
Cronk, L. B., with Brown, deSinner, Green, Gibbons & Kuitunen-Ekbaum, 558 (Hel.)
Cros, R., with Gallais & Capponi, 7 (Mal.)
Cross, H. F. & Fye, R., 784 (Ent.)
Crowell, R. L., with Hess, 8 (Mal.)
Cruickshank, R., 837 (Typh.)
Cruz, W. O., 717 (Mal.)
Cuba, 509 (B. R.)
Culbertson, C. G., with Powell, 733 (Rab.)
Cullumbine, H., (158) (Heat Str.)
——, with Bibile, Kirtisinghe, Watson & Wikramanayake, (87) (Misc. Dis.)
——, with ——, ——, Wickramanayake & Watson, (77) (Haem.)
——, with ——, Watson & Wikramanayake, 168 (Reports, etc.)
——, Bibile, S. W. & Wikramanayake, T., (1216) (Def. Dis.)
Culwell, W. B., with Coatney, Cooper, White & Imboden, 1177 (Mal.)
——, with ——, ——, ——, Lints & Eyles, 1060 (Mal.)
——, with Cooper, Coatney, Eyles & Young, 1172 (Mal.)
——, with Lints, Coatney, Cooper, White & Eyles, 1059 (Mal.)
Culwick, A. T., with Fairbairn, 605, 820, 1186 (Tryp.)
Cunha, J. P., with Marsiaj, Guimarães & Lima, 399 (Der.)
Curbelo Hernandez, A., (543) (R.F.)
Curd, F. H. S., with Birtwell & Rose, (205) (Mal.)
—— & Davey, D. G., 827 (Tryp.)
——, ——, Hendry, J. A. & Rose, F. L., 1185 (Mal.)
Curia, L., with Quiroga & Ursi, (457) (Leish.)
Cuthbertson, W. F. J., Lloyd, J. F., Emery, W. B. & Lees, K. A., (77) (Haem.)
Cutileiro, J., 1191 (Typh.)
Czuczwar, S., with Krynski, 126 (Typh.)

D

Dähne, G., 202 (Mal.)
Dailey, H. R., with Mantz & Grocott, 406 (Prot.)
Dakshinamurty, S., 1052 (Mal.)
Dalip Singh, with Jaswant Singh, 963 (Mal.)
Dalmat, H. T., 895 (Ent.)
Dalton, H. C., with Hunter & Dillahunt, 10 (Hel.)
Damasceno, R. G., Arouck, R. & Causey, O. R 1231 (Ent.)
——, with Deane, 710 (Mal.)
Damasceno, R. M. G., with Deane, (1188) (Tryp
van Damme, R., with van Grembergen Vercruysse, (1211) (Hel.)
Dana, R., Sebag, A., Cohen, J. & Borsoni, C 1074 (Typh.)
Danaraj, T. J., 394 (Def. Dis.)
Dantas, A., 152 (Def. Dis.)
Darrow, E. M., 323 (Mal.)
Darwis, A., with Wiradikarta, 49 (Lep.)
Darwish, A. E., 47 (R.F.)
Das, N. & Mukherji, M., (261) (Hel.)
Dasgupta, B. K., (112) (Mal.)
Das Gupta, P. R., with Kapur, 592 (Mal.)
Dauis-Lawas, D. F., with Stransky & Vicent 1217 (Def. Dis.)
Dave, C. J., (864) (Hel.)
Davey, D. G., 968 (Tryp.)
——, with Curd, 827 (Tryp.)
——, with ——, Hendry & Rose, 1185 (Mal.)
Davey, J. T., with Nash, 718 (Tryp.)
David, A., with Singh, 1169 (Mal.)
David, W. A. L., 90 (Ent.)
—— & Gardiner, B. O. C., (784) (Ent.)
—— & Tew, R. P., (Ent.)
Davidovici, S., with Mer, 567 (Ent.)
Davidson, G., 597, 959 (Mal.)
Davidson, L. S. P. & Fountain, J. R., 1027 (Sp.)
Davies, E. R. & Stewart, I. S., (759) (Hel.)
Davies, J. N. P., 85 (Misc. Dis.), 526 (Tryp.)
Davis, A. B., with Laird & Drinnon, 743 (Am.)
Davis, A. K., with Overman, Bass & Golde 116 (Mal.)
Davis, A. N., Jr., with Deonier, Raun, Peek Nottingham, 443 (Mal.)
Davis, A. T., with Kirk & Haseeb, 1193 (Rab.)
Davis, D. E., with Yeh, 621 (Pl.)
Davis, D. H. S., 354 (Pl.)
Davis, D. J., with Sullivan, McGregor & Eac 455 (Tryp.)
Davis, E. D., with Raley, 95 (Ent.)
——, with Rees & Raley, 163 (Ent.)
Davis, T. R. A., 64 (Hel.)
Davison, A. R., 471 (Lep.)
Dawood, M., with Halawani & Baz, 64 (Hel.)
Dawood, M. M., with Halawani, 1204 (Hel.)
Dawson, C. E. & Blagg, W., 739 (Chl.)
Dawson, K. E., Sherman, A. J. & LaBoccet A. C., 566 (Ent.)
De, U. N., with Chakravarty, Sen Gupta & Bo 726 (Leish.)
Deane, L. M. & Damasceno, R. G., 710 (Mal.)
——, Lêdo, J. F., Freire, E. P. S., Contrim, Sutter, V. A. & de Andrade, G. C., 1065 (Ma
—— & Sutter, V. A., 1058 (Mal.)
Deane, M. & da Costa, O. R., 769 (Hel.)
Deane, M. P., 609 (Tryp.), 1212 *bis* (Hel.)
—— & Damasceno, R. M. G., (1188) (Tryp.)
DeBoer, C. J., with LeBell, Hazz & Cox, 8 (Rab.)
Decourt, P., 302 (Mal.)
——, Dupoux, R. & Pelloux, A., 140 (Hel.)
——, with Schneider & Montézin, 325 (Mal.)
Decoussau, D., with Hoxter, (78) (Vms.)

Deeb, A. A., with Erfan, 253 (Hel.)
Dejou, L., 260 (Hel.)
DeLamater, J. N., with Hallman & Michaelson, 1086 (Am.)
DeLeon, J. F., with Salcedo, Pedroche & Panganiban, 73 (Def. Dis.)
Dell'Aquila, A., (539) (Am.)
Del Pozo, E. C., 489 (Vms.)
—— & Derbez M., J., 270 (Vms.)
Del Rosario, F. G., with Smith & Dy, (1170) (Mal.), 1234 (Ent.)
DeMarsh, Q. B., with Boyce, S., 1218 (Haem.)
Dempster, G. O. L., 753 (Lep.)
Dengler, H. P., (715) (Mal.)
Denhoff, E. & Laufer, M. W., 150 (Hel.)
Deniel, H., with Floch & Destombes, (1100), 1100 (Lep.)
Dennis, E. W., 981 (Am.)
——, with Berberian, 114 (Mal.)
——, Berberian, D. A. & Hansen, S. S., 243 (Am.)
Denoix, P., with Maujol, L. & Moine, M., 100 (Reports, etc.)
Denoix, P. F., 1223 (Misc. Dis.)
Deom, J., 969 (Tryp.)
Deonier, C. C., with Fluno, Raun & Faulkner, 568 (Ent.)
——, with Incho, 785 (Ent.)
——, Raun, E. S., Peek, E. H., Davis, A. N., Jr. & Nottingham, E., 443 (Mal.)
——, Sullivan, W. N., Nottingham, E. & Lindquist, A. W., 13 (Mal.)
Derbez M., J., with Del Pozo, 270 (Vms.)
Desai, S. D., with Bhende, Purandare, Banker & Figueredo, 337 (Leish.)
Deschamps, G., with Beaver, 849 *bis* (Am.)
Deschiens, R., Bablet, J. & Lambault, E., 481 (Hel.)
—— & Pick, F., 16 (Mal.)
deSinner, F., with Brown, Cronk, Green, Gibbons & Kuitunen-Ekbaum, 558 (Hel.)
Destaing, F., with Benhamou, Albou & Pugliese, 830 (Leish.)
Destombes, P., with Floch, 548, 550, 551, 757 *bis*, 1095, 1099, 1100, 1101, (1101) *bis* (Lep.)
——, with —— & Deniel, (1100), 1100 (Lep.)
Deutsch, D. L. & Peterson, E. T., 840 (Typh.)
Devignat, R., 536 (Pl.)
——, with Wolfs, 637 (Hel.)
Dey, N. C., with Ghosh & Panja, (1221) (Der.)
Dharmendra, 367, 632 (Lep.)
—— & Mukerjee, 367 (Lep.)
—— & Mukherjee, N., 755 (Lep.)
—— & Sen, N., 368 (Lep.)
Diacos, C., 395 (Def. Dis.)
Diallo, with Schneider, Marchalant, Lotte & Buisson, 449 (Tryp.)
Dias, C. B., 177 (B.R.)
Dias, E., 123, 217 (Tryp.)
——, with Laranja & Nobrega, 22 (Tryp.)
Diaz-Calderin, R., with Rodriguez-Molina, Acevedo, Torres, Lopez-Sanabria & Ramirez-Rodriguez, 1206 (Hel.)
Diaz Marin, J. & Irigoyen Ramirez, A. I., 854 (R.F.)
Diaz Melchor, F. & Aparicio Garrido, J., 717 (Mal.)
Díaz Nájera, A., with Vargas, 669 (Ent.)
Diaz Sanchez, M., (805) (Mal.)
Dick, G. W. A., with Smithburn, Goodner, Kitchen & Ross, 229 (Y.F.)
Dickson, R. C. & Ross, M., 37 (Am.)
Diercks, F. H., with Smadel & Bailey, 616 (Typh.)
——, with ——, Traub, Frick & Bailey, 615 (Typh.)
——, with Traub & Frick, 1077 (Typh.)
Diez Melchor, F., (1201) (Am.)
Dillahunt, J. A., with Hunter & Dalton, 1007 (Hel.)
Dinnik, J. & Dinnik, N., (390) (Hel.)
Dinnik, J. A., 640 (Hel.)
Dinnik, N., with Dinnik, (390) (Hel.)
Dixit, D. T., with Subramanian, 431 (Mal.)
Dixon, D. S., 807 (Mal.)
Djoa Liang Ham, with Bras, Rijkebüsch & Kotter, 83 (Der.)
Doane, J. C., with Blumberg & Weiss, (27) (Typh.)
——, with Murray, Baehr, Shwartzman, Mandelbaum, Rosenthal, Weiss, Cohen & Snyder, 729 (Typh.)
Doddananjayya, R., 130 (Typh.)
Doetschman, W. H. & Furman, D.P., 161 (Ent.)
Dogra, J. R., 33, 980 (Rab.)
——, with Rao, 33 (Rab.)
Dominguez, A. & Dominguez, M., (803) (Mal.)
Dominguez, E., with Pifano, Mayer, Römer, Medina & Benaim Pinto, 723 (Tryp.)
Dominguez, M., with Dominguez, (803) (Mal.)
Donaldson, A. W., with Brooke, 518 (Mal.)
Donaldson, R., with King, Gilchrist, Wootton, Sisson, Macfarlane, Jope, O'Brien, Peterson & Strangeways, 152 (Haem.)
Donckaster, R., with Neghme & Silva, (1107) (Hel.)
Donzelot, E. & Kaufmann, H., 613 (Typh.)
——, —— & Vernant, P., 971 (Typh.)
Dooling, H. C., 99 (Reports, etc.)
Doorenbos, W. & Cossery, G. N., (739) (Chl.)
——, with ——, (739) (Chl.)
Do Páteo, J. D., Jr., 999 (Lep.)
Do Prado, F. C., 626 (Am.)
Dorer, R. E., (715) (Mal.)
Dossena, P., 269 (Vms.)
Dostrovsky, A. & Ticho, A., 374 (Lep.)
Doughty, J. M., Jr., (733) (Rab.)
Dowling, M. A. C., 321 (Mal.)
Downs, W. G., 107, 587 (Mal.)
—— & Bordas, E., 429, 587 (Mal.)
Drayer Brito, A., with Benaim Pinto, 721 (Tryp.)
Drinnon, V. P., with Dubin & Laird, 327 (Mal.)
——, with Laird & Davis, 743 (Am.)
Driver, M. W., with Humphreys, Campbell & Hatton, 751 (R.F.)
Duarte, E., with Torres, 336 (Tryp.)
Dubin, I. N., Laird, R. L. & Drinnon, V. P., 327 (Mal.)
Dubois, A., 540 (R.F.)
Duc, N., with Harant, 510 (B.R.)
Dudgeon, J. A., with Cathie, 496 (Prot.)
Duemling, W. W., (271) (Der.)
Duguet, J., 410 (Ent.)
Dumoulin, F. V. B., 887 (Misc. Dis.)
Dunn, M. C., with Thompson, Bayles & Reinertson, 741 (Am.)
——, with —— & Winder, 750 (R.F.)
Dupoux, R., with Decourt & Pelloux, 140 (Hel.)
——, with Durand & Schneider, 957 (Mal.)

Durand, P., Schneider, J. & Dupoux, R., 957 (Mal.)
Dwivedi, J. K., 34 (Rab.)
Dworetzky, M., with Bouquet & Essex, (1030) (Vms.)
Dy, F. J., 301 (Mal.)
——, Bernardo, B. C. & Bernardino, C. E., 1178 (Mal.)
——, with Smith, 211 (Mal.)
——, with —— & Cabrera, 1173, 1174 (Mal.)
——, with —— & Del Rosario, (1170) (Mal.), 1234 (Ent.)

E

Eads, R. B., Henderson, H. E., McGregor, T. & Irons, J. V., 470 (R.F.)
——, with Sullivan, McGregor & Davis, 455 (Tryp.)
Eagle, H., with Germuth & Oyama, 1188, 1189 (Leish.)
Earle, A. M., with Reilly, 1191 (Typh.)
Earle, K. V., 81 (Der.), 850 (Am.)
East Africa High Commission, 819 (Tryp.)
East African Med. J., 266, 488 (Haem.), 526 (Tryp.)
Eastwood, C. G., 1073 (Tryp.)
Ecker, H. D., Ambury, J. F. & Robinson, L. B., 124 (Leish.)
Eckstein, A., (711) (Mal.)
Edelman, M. H. & Spingarn, C. L., 56 (Hel.)
Edelstein, E., with Krakowsky, (469) (R.F.)
Edeson, J. F. B. & Field, J. W., 437 (Mal.)
——, with ——, 12 (Mal.)
Edwards, J. C., 231 (Y.F.)
Eeraerts, W., with Reul, 31 (Den.)
Ehlers, H. J. & Knüttgen, H., 638 (Hel.)
Eichenwald, H., 159 (Prot.)
Eichholtz, F., Hotovy, R., Sauer, A. & Weissflug, I., 1025 (Hel.)
Eichler, W. & Pagast, F., 431 (Mal.)
El Din Nor El Din, G., 1088 (Am.)
Elerding, A. C., with Talbot & Westwater, 591 (Mal.)
Ellerman, J. R., 180 (B.R.)
Elliott, D. C., 1096 (Lep.)
Ellis, F. P., (1031) (Heat Str.)
Elmore, F. H., with Rosenthal, 776, 881 (Der.)
Elsdon-Dew, R., 538 (Am.)
——, with Armstrong & Marot, 628 (Am.)
——, with —— & Wilmot, 994 (Am.)
Elson, B. E., with Jellison, Welsh & Huebner, 731 (Typh.)
Elton, N. W., with Butz, (1226) (Misc. Dis.)
—— & Johnson, C. M., 732 (Y.F.)
Emerson, G. A., with Parker & Ewing, 969 (Tryp.)
Emery, W. B., with Cuthbertson, Lloyd & Lees, (77) (Haem.)
Emmel, L., 254 (Hel.)
Emmett, J., 135 (Am.), 608 (Tryp.)
Emmons, C. W., 659 (Der.)
——, Morlan, H. B. & Hill, E. L., 491 (Der.)
Eneboe, P. L., with Jellison, Parker & Hughes, 470 (R.F.)
Engler, G. & Sanchez, G., 484 (Hel.)
Enigk, K., (552) (Hel.), (570) (Ent.)
Erfan, H. & Deeb, A. A., 253 (Hel.)
Erfan, M., 633 (Hel.)
Erickson, P. T., with Johansen & Ross, 547 (Lep
——, with ——, Wolcott, Meyer, Gray, Preje & Ross, 550 (Lep.)
Eskridge, M., with Peake, 745 (Am.)
Espin, J., with Vegas & Convit, 631 (Lep.)
——, with Vogelsang, 771 (Hel.)
Espinoza, J., with Meneghello & Coronel, (Def. Dis.)
Esquivel, R. B., with Boccalandro, 352 (Rab.)
Essex, H. E., with Boquet & Dworetzky, (103 (Vms.)
Eucker, H., 557 (Hel.)
Ewing, P. L., with Parker & Emerson, 969 (Tryp
Eyles, D., with Coatney, Cooper, White, Lints Culwell, 1060 (Mal.)
Eyles, D. E., with Cooper, Coatney, Culwell Young, 1172 (Mal.)
—— & Jeffery, G. M., 306 (Mal.)
——, with Lints, Coatney, Cooper, Culwell White, 1059 (Mal.)
——, with Young, 304 (Mal.)

F

Fabregat, with Harant, 864 (Hel.)
Fabregoule, with Thiodet, 204 (Mal.)
Faiguenbaum, J., Agosin, M. & Tamargo, A (1106) (Hel.)
Fain, A., with Jadin, 431 (Mal.)
Fairbairn, H. & Culwick, A. T., 605, 820, 118 (Tryp.)
Fairbairn, J. W. & Lou, T. C., (522) (Mal.)
Fairley, N. H., 3 (Mal.)
——, with Shortt, Covell, Shute & Garnham, 19 (Mal.)
Falconí Villagómez, J. A., (521) (Mal.)
Fallas, M., with Romero & Ibarra, 371 (Lep.)
de Faria, J. L., 248, (1095) (Lep.)
Farquhar, H. G. & Turner, W. M. L., 89 (Prot
Faulkner, F., with Fluno, Raun & Deonier, 56 (Ent.)
Fauré, J., with Gaud & Maurice, 803 (Mal.)
Faust, E. C., (564) (Prot.)
——, with Carrera, 465 (Am.)
——, with Fuller, 464 (Am.)
Fauvert, R., with Bertrand-Fontaine, Schneider & Hartmann, 433 (Mal.)
Favarel, R., 236 (Pl.)
Fay, R. W., Baker, W. C. & Grainger, M. M 94 (Ent.)
—— & Sheppard, E. H., 95 (Ent.)
Febres Cordero, R., with Potenza & Anduze, 77 (Hel.)
Feder, A., 435 (Mal.)
Felix, A., 837, 971, (Typh.)
Fendall, N. R. E., 844 (Rab.)
Feng, L. C., Lin, C., Ting, H. C. & Hwang, J. C. 144 (Hel.)
Feng, L. P., with Hsu & Chang, 952 (Mal.)
Feng, T., 737 (Pl.)
Ferguson, A. L., 537 (Pl.)
Ferguson, F. F., with Mathis, Upholt & Quarter man, 892 (Ent.)
Fernandez Baquero, G., with Gonzalez Periz & Fornos Palencia, (667) (Misc. Dis.)
Fernando, C. F., 238 (Am.)
Feron, J., (1101) (Lep.)
Ferrada-Urzúa, L. V., with Mackinnon & Monte mayor, 490 (Der.)

Ferrand, J., with Garcin, 663 (Oph.)
Ferreira, F. C. & Pinto, A. R., 998 (Y.S.)
Ferreira, F. S. da C., 966, 1069 (Tryp.)
—— & de Almeida, C. L., 967 (Tryp.)
—— & Pinto, A. R., 1202 (Ys.)
Ferreira, M., with Lacaz, Iaria, Martins & Vega, 494 (Der.)
Ferreira, M. O. & Rachou, R. G., 212 (Mal.)
——, with ——, 815 (Mal.)
——. with —— & de Bustamante, 600 (Mal.)
Ferro-Luzzi, G., 57, 639 (Hel.)
Fessler, K., Fischer, L. & Sorg, G., 1169 (Mal.)
Field, J. W., 8, 307 (Mal.), 785 (Ent.)
—— & Edeson, J. F. B., 12 (Mal.)
——, with ——, 437 (Mal.)
Fiennes, R. N. T.-W., 827 (Tryp.)
Figueredo, N., with Bhende, Purandare, Banker & Desai, 337 (Leish.)
Figueroa, J., 735 (Rab.)
Files, V. S. & Cram, E. B., 376 (Hel.)
Filho, J. C. A., with de Bustamante & Aguirre, 815 (Mal.)
Findlay, G. M., 13 (Mal.), 329 *bis* (Bl.), 837 (Typh.), 905 (B.R.)
——, with Ampofo, 631 (Ys.)
Fischer, L., with Fessler & Sorg, 1169 (Mal.)
Fischer, O., 309, 313, 319 (Mal.)
—— & Krause, G., 339 (Leish.)
Fischl, M., 233 (Rab.)
Fishman, A. P. & Kinsman, J. M., 203 (Mal.)
Fitzgerald, M. E. H., with Collier & Campbell, 980 (Chl.)
Flamm, H., 773 (Sp.)
Flecker, H., with Reid, 564 (Vms.)
Fleming, A., 837 (Typh.)
Fletcher, O. K., Jr., with Beaver, (407) (Ent.)
Floch, H., (247), 1093 (Lep.), 713 (Mal.)
—— & Abonnenc, E., 121, (219), 219, (529) *bis* (Tryp.)
——, Deniel, H. & Destombes, P., (1100), 1100 (Lep.)
—— & Destombes, P., 548, 550, 551, 757 *bis*, 1095, 1099, 1100, 1101, (1101) *bis* (Lep.)
Flores Navarro, J., with Barba Rubio & Aceves Ortega, 758 (Lep.)
Florey, H., 837 (Typh.)
Florio, L., with Koprowski, Cox & Miller, 1080 (Den.)
——, Miller, M. S. & Mugrage, E. R., 978 *bis* (Den.)
de Fluiter, H. J., 709 (Mal.)
Fluno, J. A., Raun, E. S., Deonier, C. C. & Faulkner, F., 568 (Ent.)
Fogg, V. H., with Tomlinson & Smith, 160 (Prot.)
Fonseca, F., Nunes, M. A., Pinto, M. R. & Lacerda, M. T., 532 (Typh.)
——, Pinto, M. R., de Azevedo, J. F., Amaro, F. C. & Lacerda, M. T., 532 (Typh.)
——, ——, —— & Lacerda, M. T., 532 (Typh.)
——, ——, Gander, G., de Azevedo, J. F. & Lacerda, M. T., 532 (Typh.)
——, ——, Oliveira, J., da Gama, M. M. & Lacerda, M. T., 532 (Typh.)
Forbes, J., (1077) (Typh.)
Fornos Palencia, P., with Gonzalez Peris & Fernandez Baquero, (667) (Misc. Dis.)
Fort, M., with Schwetz & Baumann, 191 (Mal.)
Forteza Bover, J., Garrigues Orellana, A. & Marco Orts, F., 854 (R.F.)
Fossen, A., with Hausman & Yoe Tjin Liong, 766 (Hel.)
Fountain, J. R., with Davidson, 1027 (Sp.)
Fouquet, J. & Morin, M., 841 (Typh.)
Fournier, J. & Lieou, Y. C. (980) (Chl.)
Fourrier, with Babeau, 330 (Bl.)
——, with Thiodet, 110 (Mal.)
Fourrier, A., with Thiodet, 315, 316 (Mal.)
——, with ——, Boulard & Raffi, 111 (Mal.)
Fox, E. C., 79 (Der.)
Fox, H. J., 74, 880 (Sp.)
Fox, I. & Kohler, C. E., 783 (Ent.)
Fox, J. P., 27 (Typh.)
Franco, R., 615 (Typh.)
Frappier, A., with Guy & Panisset, 82 (Der.)
Fraser, N. D., 473 (Lep.)
Frazer, A. C., 74, 75 (Sp.)
Frazier, C. A. & Rice, C. E., 1122 (Haem.)
Freeman, P., 895, 896, (1130) (Ent.)
—— & Pryor, H. B., (1229) (Prot.)
Freeman, R. B., (669) (Ent.)
Freire, E. P. S., with Deane, Lédo, Cotrim, Sutter & de Andrade, 1065 (Mal.)
Freire, S. A., with de Magalhães & Rocha, 839 (Typh.)
Freund, J., with Lipton, 845 (Rab.)
Freygang, F., 346 (Typh.)
Frick, L. P., with Smadel, Traub, Diercks & Bailey, 615 (Typh.)
——, with Traub, 616 (Typh.)
——, with —— & Diercks, 1077 (Typh.)
Friess, J., with Marill, 253 (Hel.)
Frohne, W. C., Weathersbee, A. A., Williams, G. M. & Hart, J. W., 709 (Mal.)
Frost, S. W., 953 (Mal.)
Fry, N. H., with Hunter, Bennett, See & Greene, 380 (Hel.)
Fukuzumi, S., Itoh, H., Yamashina, A. & Ohwada, H., 729 (Typh.)
Fuller, F. W. & Faust, E. C., 464 (Am.)
Fuller, H. S., Murray, E. S. & Snyder, J. C., 127 (Typh.)
Fulton, J. D., 121 (Tryp.)
—— & Joyner, L. P., 529 (Leish.)
——, —— & Chandler, R. L., 1188 (Leish.)
Fulton, R. A., with McBride & Sullivan, 786 (Ent.)
Furman, D. P., with Doetschman, 161 (Ent.)
—— & Kurtpinar, H., 1123 (Vms.)
Fye, R., with Cross, 784 (Ent.)

G

Gaase, A., 559, 879 (Hel.)
Gabaldon, A., 4 (Mal.)
Gale, G. W., 281 (Reports, etc.)
Gallais, P., Cros, R. & Capponi, M., 7 (Mal.)
Gállego Berenguer, J., with Covaleda Ortega, (965) (Mal.)
Galley, R. A. E., with Whittingham, 93 (Ent.)
Galliard, H., 145, (1018) (Hel.)
—— & Brumpt, L. C., 58 (Hel.)
——, —— & Martinez, R., 888 (Misc. Dis.)
——, Grenierboley, J. & Phiem, N. H., (1040) (Ent.)
——, with Huard & Joyeux, (768) (Hel.)
—— & Lapierre, J., 1182, 1183 (Mal.)
——, Mille, R. & Robinson, W. A., 146 (Hel.)
——, —— & Robinson, W. H., 60 (Hel.)
—— & Ngu, D. V., (565) (Ent.)
—— & Ribo, (1040) (Ent.)

Galvão, A. B., 710 (Mal.)
da Gama, M. M., with Fonseca, Pinto, Oliveira & Lacerda, 532 (Typh.)
Gambini, G., (156) (Der.), 780 (Misc. Dis.)
——, with Amalfitano, Benetazzo, Cifarelli, Servino & Tarizzo, 123 (Leish.)
Gan, K. H., (469) (Am.)
Gander, G., with Fonseca, Pinto, de Azevedo & Lacerda, 532 (Typh.)
Gander, R., with Geigy, 498 (Ent.)
Ganguli, N., with Adhikari, 1181 (Mal.)
Garabedian, G. A., with Azar & Pipkin, 141 (Hel.)
Garcia Lopez, G., Milanes, F., Spies, T. D., Lopez Toca, R., Aramburu, T. & Lopez, H., 396 (Sp.)
García–Zorron, N., with Mackinnon, 1125 (Der.)
Garcin, G. & Ferrand, J., 663 (Oph.)
Gard, S., Magnusson, S. H., Wahlgren, F. & Gille, G., 1039 (Prot.)
Gardiner, B. O. C., with David, (784) (Ent.)
Gardner, H. T., with Burlingame, Resemann, Cranmer & Vollmer, 358 (Am.)
Garnham, P. C. C., 4, 707, 1067 (Mal.), 354 (Pl.), 1129 (Prot.)
——, with Shortt, Fairley, Covell & Shute, 193 (Mal.)
——, with Wilson & Swellengrebel, 677 (Mal.)
Garrett–Jones, C., 583 (Mal.), 782 (Ent.)
Garrigues Orellana, A., with Forteza Bover & Marco Orts, 854 (R.F.)
Gasq, 447 (Tryp.)
Gasq, M., 821, 967 (Tryp.)
—— & Lapeyssonnie, L., 821 (Tryp.)
Gatti, J. C., with Basombrio, 754 (Lep.)
Gaud, J., Fauré, J. & Maurice, A., 803 (Mal.)
——, Méchali, D. & Clier, J. L., 595 (Mal.)
——, Schneider, J. & Mechali, D., 1176 (Mal.)
Gaud, M., 351 (Typh.)
Gaul, A. T., 155 (Vms.)
Gauld, R. L., with Bennett & Smadel, 223 (Typh.)
——, with Berge & Kitaoka, 349 (Typh.)
——, with Crocker, Bennett, Jackson, Snyder, Smadel & Gordon, 617 (Typh.)
Gay, B., with de Prada & Llorente, 533 (Typh.)
Gear, J. H. S., Wolstenholme, B. & Cort, A., 977 (Typh.)
Gehr, E. & Gehr, E., 859 (Lep.)
Gehr, E., with Gehr, 859 (Lep.)
Geib, A. F., 715 (Mal.)
——, with Smith, 277 (Ent.)
Geigy, R. & Gander, R., 498 (Ent.)
—— & Rahm, U., 15 (Mal.)
Geiling, E. M. K., with Reilly & Chen, 326, 717 (Mal.)
Geiman, Q. M. & McKee, R. W., 1036 (Prot.)
Gelfand, M., 376 (Hel.), 404, 405, 779 (Misc. Dis.), 903 (B.R.)
Gelsi, R. & Lacroix, A., (958) (Mal.)
Gemar, F., with Ross, 1000 (Lep.)
Genú, J. O. Á. & de Oliveira Lima, S., 551 (Lep.)
——, —— & Santos, M., 551 (Lep.)
Gerichter, C., with Yoeli, Steinitz & Rabinowicz, 37 (Am.)
Gerichter, C. B., with Silberstein & Rabinowitz, 234 (Rab.)
Gerloff, R. K., with Lackman & Parker, 224 (Typh.)
Germain, A., André, L. & Marty, J., 1113 (Hel.)
Germer, W. D. & Heni, F., 460 (Typh.)
Germuth, F. G., Jr., Eagle, H. & Oyama, V., 1188, 1189 (Leish).
Geukens, 949 (Mal.)
Ghanem, M. D., with Salah, 87 (Misc. Dis.)
Ghosh, B. N., (1123) (Vms.)
Ghosh, H., Ghosh, N. N. & Ray, J. C., 829 (Leish.)
Ghosh, L. M., 48 (Lep.)
——, Dey, N. C. & Panja, D., (1221) (Der.)
Ghosh, N. N., with Ghosh & Ray, 829 (Leish.)
Ghosh, S. M., 896 (Ent.)
Ghosh, T. N., with Bose & Bose, 1198 (Am.)
Giacobbe, O., with Vucetich, 150 (Hel.)
Gibbons, J. E., with Brown, Cronk, deSinner, Green & Kuitunen-Ekbaum, 558 (Hel.)
Gibson, J. B. G., 664 (Oph.)
Gilbertson, W. E., with Andrews, 815 (Mal.)
Gilchrist, M., with King, Wootton, Donaldson, Sisson, Macfarlane, Jope, O'Brien, Peterson & Strangeways, 152 (Haem.)
Gilchrist, W. S., 542 (R.F.)
Gil Collado, J., (897) (Ent.)
Giles, H. McC. & Symington, T., 348 (Typh.)
Gille, G., with Gard, Magnusson & Wahlgren, 1039 (Prot.)
Gillet, J., 632 (Hel.)
Gillett, J. D., with Haddow, Mahaffy & Highton, 1231 (Ent.)
——, with Smithburn & Haddow, (32) (Den.)
Gillette, H. P. S., with Newbold, 809 (Mal.)
Gillman, C., 215 (Tryp.)
Gilroy, J. C., 396 (Sp.)
Gimeno de Sande, A., 834, 835, (835) (Typh.), 855 (R.F.)
Ginés, A. R., 257 (Hel.)
——, Gould, E. & Melgarejo de Talavera, S., 400 (Der.)
Giordano, M., 1238 (B.R.)
Girard, G., 236 (Pl.)
Girdwood, R. H., (774) (Sp.), 789 (Reports, etc.)
Giroud, P. & Ciaccio, G., 613 (Typh.)
——, —— & Pan, H. S., 613 (Typh.)
——, —— & Vargues, R., 613 (Typh.)
—— & Vargues, R., 612 (Typh.)
—— & Wei, W. P., 1190 (Typh.)
Gispen, R., 615, 836 (Typh.)
——, Smit, A. M. & Westermann, C. D., (129) (Typh.)
Githens, T. S., (898) (Reports, etc.)
Gluck, V., with Campins & Scharyj, 775 (Der.)
Goble, F. C., 828 (Tryp.)
Godbole, S. H., Ramachandra Rao, T. & Viswanathan, D. K., (950) (Mal.)
Godfrey-Horan, M. E., with Sloan, Chung-Hoon & Hedgcock, 1098 (Lep.)
Godfried, E. G., 267, (269) (Haem.)
Godkin, S. R. S., 886 (Ulc.)
Goldacre, R. J., 671 (Ent.)
Golden, A. & Overman, R. R., 110 (Mal.)
——, with ——, Bass & Davis, 116 (Mal.)
Goldenson, J., Norton, H. E., Novak, C. F. & Sass, S., 164 (Ent.)
Goldman, M. & Johnson, S. A., 552 (Hel.)
Golyanitskaya, O. N., 382 (Hel.)
Gómez, R. F., 1124 (Der.)
Gómez Herrera, J., (712) (Mal.)

Gonçalves, J. M. & Vieira, L. G., 1122 (Vms.)
Gönnert, R., 221 (Typh.)
——, with Kikuth, 54, (637) (Hel.)
Gonnet, C. & d'Haussy, R., 1221 (Oph.)
——, with Néel & Payet, 137 (R.F.)
González Castro, J., 1004 (Hel.)
Gonzalez Peris, G., Fernandez Baquero, G. & Fornos Palencia, P., (667) (Misc. Dis.)
Gonzalez Prendes, M. A., 545 (Lep.)
—— & Ybarra Pérez, R., 138 (Lep.)
Gonzalez Robles, F., with Ortiz Mariotte, Varela & Roch, 973 (Typh.)
González Vicente, D., 1069 (Tryp.)
Goodner, K., with Smithburn, Dick, Kitchen & Ross, 229 (Y.F.)
Goodwin, L. G., 324 (Mal.)
——, with Brownlee, Goss, Woodbine & Walls, 1070 (Tryp.)
——, Goss, M. D. & Lock, J. A., 1072 (Tryp.)
——, ——, —— & Walls, L. P., 1071 (Tryp.)
Goodwin, M. B., with Hauschka, Palmquist & Brown, 607 (Tryp.)
Goodwin, M. H., Jr., 433 (Mal.)
——, with Bradley, 301 (Mal.)
Goodwine, C. H., with Boldt, 320 (Mal.)
Goor, S., 233 (Rab.)
van Goor, W. T. & Lodens, J. G., 807 (Mal.)
Gordon, M. K., with Crocker, Bennett, Jackson, Snyder, Smadel & Gauld, 617 (Typh.)
Gordon, R. M., 707 (Mal.)
——, with Crewe, 489 (Vms.)
—— & Kershaw, W. E., 1214 (Hel.)
——, ——, Crewe, W. & Oldroyd, H., 1214 (Hel.)
Gorgas Memorial Institute, 1137 (Reports, etc.)
Gorirossi, F. E., (1131) (Ent.)
Goss, M. D., with Brownlee, Goodwin, Woodbine & Walls, 1070 (Tryp.)
——, with Goodwin & Lock, 1072 (Tryp.)
——, with ——, Lock & Walls, 1071 (Tryp.)
Gougerot, H., 370 (Lep.)
Gould, E., with Ginés & Melgarejo de Talavera, 400 (Der.)
Gour, K. N., 44 (Am.)
Gowen, L. F., with Carson & Cochrane, 225 (Typh.)
Goyal, R. K., 33 (Rab.), 78 (Vms.)
de Graaf, W., 314 (Mal.)
Graeve, K. & Herrnring, G., 391 (Hel.)
Graham, J. G. & Orr, J. L., 763 (Hel.)
Grainger, M. M., with Fay & Baker, 94 (Ent.)
Gramberg, K. P. C. A., 139 (Lep.)
Gray, H. H., with Johansen, Erickson, Wolcott, Meyer, Prejean & Ross, 550 (Lep.)
Green, A. A., with Higgins, 163 (Ent.)
Green, J. E., with Brown, Cronk, deSinner, Gibbons & Kuitunen-Ekbaum, 558 (Hel.)
Green, R. & Mankikar, D. S., 658 (Der.)
Green, R. H., with Colbert & Strauss, 778 (Der.)
Greenberg, J., 214, 443 (Mal.), 407 (Ent.)
——, with Coatney, Cooper & Trembley, 444 (Mal.)
—— & Richeson, E. M., 1186 (Mal.)
——, Trembley, H. L. & Coatney, G. R., 603 (Mal.)
Greene, E., with Hunter, Bennett, Fry & See, 380 (Hel.)
Greenwood, M., 35 (Chl.)
van Grembergen, G., (1208) (Hel.)
——, van Damme, R. & Vercruysse, R., (1211) (Hel.)
Grenier, P., (669) (Ent.)
—— & Rageau, J., 669 (Ent.)
Grenierboley, J., with Galliard & Phiem, (1040) (Ent.)
Gridgeman, N. T., (165) (Lab.)
Griebel, C., 887 (Misc. Dis.)
Grieff, D., with Laskowski & Pinkerton, (1192) (Bart.)
Griffin, A. M. & McCarten, W. G., 852, 1087 *bis* (Am.)
—— & Michini, L. J., 1087 (Am.)
Grjebine, A., with Maillot, 668 (Ent.)
Grober, J., 884 (Heat Str.)
Grocott, R. G., with Mantz & Dailey, 406 (Prot.)
Groot, H., 720 (Tryp.)
Gross, B. & Bonnet, D. D., 461 (Pl.)
Grossman, E. B., with Most, Miller & Conan, 1089 (Am.)
Grossman, E. J., with Most & Miller, 1200 (Am.)
Grove-White, R. J., (364) (Lep.)
Grycz, E., 843 (Rab.)
Guérin, P., with Ravina, Pécher & Clavel, (989) (Am.)
Guerra Valdés, R., with Basnuevo, Gutiérrez Estarlí & Sánchez Beltran, (1200) (Am.)
Guerrieri, P., 582 (Mal.)
Guignard, J., with Bonnin & Morotti, 742 *ter.* (Am.)
Guilhaumou, F., 1082 (Chl.)
Guillén, J., with Contreras & Torrella, 369, 373 (Lep.)
Guimarães, F. N., 1092, 1202 (Ys.)
Guimarães, N., with Marsiaj, Cunha & Lima, 399 (Der.)
——, with Lima, (659) (Der.)
Gunn, M. A. R., 78 (Vms.)
Gupta, S. K., with Panja, 355 (Pl.)
Gurri, J., with Talice, 479 *bis* (1107) (Hel.)
Gutch, C. F., 1201 (Am.)
Gutiérrez Estarlí, E., with Basnuevo, 627, 853, (1199) (Am.)
—— with —— Guerra Valdés & Sánchez Beltran, (1200) (Am.)
Gutiérrez P., A., (745) (Am.)
Guy, R., Panisset, M. & Frappier, A., 82 (Der.)
Guyane Française & Inini, (284) (Reports, etc.), 365 (Lep.)

H

Habbu, M. K., with Sokhey, 1081 (Pl.)
Haber, L., with Pratt, Heterick & Harrison, 408 (Ent.)
Habibi, A., with Baltazard, Bahmanyar, Mofidi & Seydian, 1091 (R.F.)
——, with ——, Mofidi, Bahmanyar & Seydian, 995 (R.F.)
Hackett, C. J., 121 (Tryp.), 245 (Ys.), 790 (Reports, etc.)
Hackett, L. W., with Sasse, 1055 (Mal.)
Hadaway, A. B., 1054 (Mal.)
—— & Barlow, F., 413 (Ent.)
Haddad, E., with Scaillet, 453 (Tryp.)

Haddow, A. J., Gillett, J. D., Mahaffy, A. F. & Highton, R. B., 1231 (Ent.)
—— & Mahaffy, A. F., 230 (Y.F.)
——, with Smithburn & Gillett, (32) (Den.)
——, with —— & Lumsden, (32) (Den.)
——, with ——, Mahaffy, Kitchen & Smith, (620) (Den.)
Hadler, W. A., with Rheinboldt, Berti, Perego, Rieckmann, Rzeppa, Mauri & de Souza Lima, 548 (Lep.)
Haggard, M. E., with Schneidner & Levin, 77 (Haem.)
Hajra, B. N., 441 (Mal.)
Halawani, A., Baz, I. & Dawood, M., 64 (Hel.)
—— & Dawood, M. M., 1204 (Hel.)
Hall, D. M., Mahboob, S. & Turner, E. E., (1090) (Am.)
Hall, I. F., with Collier & Waterhouse, 236, 1196 (Chl.)
Hall, T. F. & Hess, A. D., 1061 (Mal.)
von Haller, E., 312, 711, 712 (Mal.)
Hallinan, T. C., 885 (Ulc.)
Hallman, F. A., Michaelson, J. B. & DeLamater, J. N., 1086 (Am.)
Hamers, A. L. T. M. & Hermans, E. H., 139 (Lep.)
Hamilton-Paterson, J. L., with Piney, 173 (B.R.)
Hamman, G. C., with Crawford, 661 (Oph.)
Hammick, D. L. & Mason, S. F., (437) *bis* (Mal.)
Handfield-Jones, R. P. C., 955 (Mal.)
Hanks, J. H., 1103 (Lep.)
—— & Soule, M. F., 250 (Lep.)
—— & Wallace, R. E., 368 (Lep.)
Hansen, E. L., 851 (Am.)
——, with Bradin, 538 (Am.)
Hansen, M. F., with Bern, 1052 (Mal.)
Hansen, S. S., with Dennis & Berberian, 243 (Am.)
Harant, H. & Brygoo, E., (90) (Ent.)
—— & Caron, M., 391 (Hel.)
—— & Duc, N., 510 (B.R.)
—— & Fabregat, 864 (Hel.)
—— & Théodoridès, J., 896 (Ent.)
Harding, R. D. & Hutchinson, M. P., 824 (Tryp.)
Hardy, S. M., with Hewitt, White, Hewitt, Wallace & Anduze, 1021 (Hel.)
Harinasuta, C., 991 (Am.)
Harland, J. C., Humble, J. G. & Mann, P. G., 1015 (Hel.)
Harman, J. B., 226 (Typh.)
Harper, J. O., with Heisch, 6 (Mal.)
Harrell, G. T., with Wolff, 351 (Typh.)
Harrison, J. B., with Pratt, Heterick & Haber, 408 (Ent.)
Harrison, J. L., 224, 614 (Typh.)
—— & Woodville, H. C., 1081 (Pl.)
Hart, J. T., Jr., with Mulhern, (409) (Ent.)
Hart, J. W., with Frohne, Weathersbee & Williams, 709 (Mal.)
Hartman, L., with Schneider, 311 (Mal.)
Hartman, L. W., with Sulkin & Lodowski, 888 (Prot.)
Hartmann, G., with Rissel, 863 (Hel.)
Hartmann, L., with Bertrand-Fontaine, Fauvert & Schneider, 433 (Mal.)
Hartz, P. H., 1112 (Hel.), 1224 (Misc. Dis.)
Harvey, A. E. C., with Heisch, 45 (R.F.)
Haseeb, M. A., with Kirk & Davis, 1193 (Rab.)
Haskins, W. T. & Luttermoser, G. W., 1206
——, with —— & Brady, 254 (Hel.)
Hassett, C. C. & Jenkins, D. W., 498 (Ent.)
——, with ——, 1041 (Ent.)
Hatton, G. N., with Humphreys Campbell & Driver, 751 (R.F.)
Haupt, E., (401) (Der.)
Hauschka, T. S., 335 (Tryp.)
——, Goodwin, M. B., Palmquist, J. & Brown, E. 607 (Tryp.)
Hausman, R. & Hiemstra, S., 491 (Der.)
—— Yoe Tjin Liong & Fossen, A., (766) (Hel.)
Hauss, W. H. & Simrock, W., 348 (Typh.)
d'Haussy, R., with Gonnet, 1221 (Oph.)
Havens, F. Z., Jr. & Stickney, J. M., (977) (Typh.)
Hawking, F., 1214 (Hel.)
——, with Sewell, 1023 (Hel.)
——, Sewell, P. & Thurston, J. P., 1019 (Hel.)
Hawksley, J. C. & Stokes, E. J., 970 (Typh.)
Haworth, R. D., McKenna, J. & Whitfield, G. H. (360) (Am.)
Hayes, W. J., Jr. & Simmons, S. W., (1132) (Ent.)
——, with Skaliy, 409 (Ent.)
Hayman, J. M., Jr., with Most, Kane, Lavietes, Schroeder, Behm, Blum & Katzin, 1009 (Hel.)
Hayman, R. W. & Holt, G. W. C., 180 (B.R.)
Hazato, H., with Tamiya, Yamamoto, Shimojo & Nishioka, 344 (Typh.)
Hazz, E. K., with LeBell, DeBoer & Cox, 845 (Rab.)
Head, J. A., with Conan & Brewer, 992 (Am.)
Heckenroth, F., Bécuwe, R., Mayan, L. & Leroux, G., 1114 (Hel.)
Hedgcock, G. H., with Sloan, Chung-Hoon & Godfrey-Horan, 1098 (Lep.)
van Heerdt, P. F., (565) (Ent.)
Heilbrunn, A., 43 (Am.)
Heilesen, B., 497 (Ent.)
Heilmann, C. G., 649 (Hel.)
Heinemann, H., 248 (Lep.)
Heisch, R. B., 543, 629 (R.F.)
—— & Harper, J. O., 6 (Mal.)
—— & Harvey, A. E. C., 45 (R.F.)
Held, J. P., with Worms & Péquignot, 971 (Typh.)
Hell, H. M., with Piringer, 979 (Rab.)
Henderson, E., with Seneca, 357 (Chl.)
Henderson, H. E., with Eads, McGregor & Irons, 470 (R.F.)
Henderson, J. M., (715), 949 (Mal.)
Henderson, R. R., van Someren, R. K. A. & Robertson, K. E. R., 977 (Typh.)
Hendricks, J. R., 651 (Hel.)
Hendrix, H., with Rodhain, 89 (Prot.)
Hendry, J. A., with Curd, Davey & Rose, 1185 (Mal.)
Heni, F., with Germer, 460 (Typh.)
Henle, W., with Sigel, Scott & Janton, 1078 (Typh.)
Hennessey, R. S. F., 526 (Tryp.)
Henrard, C. & Peel, E., 66 (Hel.)
Henry, M., 310 (Mal.)
Henschel, A., with Keys, Wells, Hoffbauer & Taylor, 519 (Mal.)
——, Taylor, H. L. & Keys, A., 519 (Mal.)
Herbert, D., 1195 (Pl.)
Hermans, E. H., with Hamers, 139 (Lep.)
Hernández Beguerie, R., with Milanés & López Toca, 487 (Sp.)
Hernández Fernández, S., with Perez Gallardo & Clavero, 840 (Typh.)

Hernández Morales, F., 255 (Hel.)
——, with Maldonado, Vélez Herrera & Thillet, 872 (Hel.)
——, with Pérez Santiago, 1199 (Am.)
——, Pratt, C. K., Oliver González, J. & Maldonado, J. F., 764 (Hel.)
——, Santiago Stevenson, D., Oliver González, J. & Maldonado, J. F., 477 (Hel.)
——, ——, Pérez Santiago, E., Oliver González, J. & Maldonado, J. F., 761 (Hel.)
—— & Stevenson, D. S., 256 (Hel.)
——, with Suarez, Spies, Perez & Benitez-Gautier, 397 (Sp.)
Hernandez-de-Paredes, C. & Paredes-Manrique, R., 724 (Tryp.)
Herrera, G., 374 (Lep.)
Herrera, L. A., with Vogelsang, (766) (Hel.)
Herrnring, G., with Graeve, 391 (Hel.)
Hertig, M., 343 (Leish.), (669) (Ent.)
Hess, A. D., 715 (Mal.)
—— & Crowell, R. L., 8 (Mal.)
——, with Hall, 1061 (Mal.)
——, with Kruse & Ludvik, 672 (Ent.)
Hesse, E., 779 (Misc. Dis.)
Heterick, R. H., with Pratt, Harrison & Haber, 408 (Ent.)
Hewitt, D. B., with Hewitt, White, Hardy, Wallace & Anduze, 1021 (Hel.)
Hewitt, R., 387 (Hel.)
——, with Briceño Rossi, 387 (Hel.)
——, Kenney, M., Chan, A. & Mohamed, H., 871 (Hel.)
——, Wallace, W. & White, E., 1198 (Am.)
Hewitt, R. I., White, E., Hewitt, D. B., Hardy, S. M., Wallace, W. S. & Anduze, R., 1021 (Hel.)
Hiemstra, S., with Hausman, 491 (Der.)
Higashi, A., with Peters & Welch, 147 (Hel.)
Higgins, A. E. H. & Green, A. A., 163 (Ent.)
Highton, R. B., with Haddow, Gillett & Mahaffy, 1231 (Ent.)
Hill, C. M., with Hill, 890 (Ent.)
Hill, E. L., with Emmons & Morlan, 491 (Der.)
——, with Morlan & Schubert, 458 (Typh.)
Hill, J. H., 143 (Hel.)
Hill, R. B., 964 (Mal.)
—— & Hill, C. M., 890 (Ent.)
Hisar, R., 275 (Misc. Dis.)
Hitchcock, D. J., 391, 862 (Hel.)
Hoare, C. A., 121 (Tryp.), 575 (B.R.), 1083 (Am.)
Hodges, J. H., 1218 (Haem.)
Hoecker, G., with Pizzi, Agosin, Christen & Neghme, 455 (Tryp.)
Hoekenga, M. T., 592 (Mal.)
—— & Box, L., 390 (Hel.)
Hoffbauer, F. W., with Keys, Wells, Taylor & Henschel, 519 (Mal.)
Hohenner, K., 374 (Lep.)
Holmes, F. O., 102 (B.R.)
Holstein, M., 584 *bis*, 803 (Mal.)
Holt, G. W. C., with Hayman, 180 (B.R.)
Holten, J. R., with Jones, (415) (Ent.)
Holtzwart, F., with Kessel & Yeaman, 82 (Der.)
Hølund, T., 1039 (Prot.)
Hopkins, G. H. E., 1142 (B.R.)
Horan, J. S., 473 (Lep.)
Horder, 173 (B.R.)
Hornby, H. E., 20 (Tryp.)
Hormann, H., 174 (B.R.), 311, 313 (Mal.)
Horne, G. O. & Mole, R. H., 80 (Der.)
Horsfall, W. R., 786 (Ent.)
Hotovy, R., with Eichholtz, Sauer & Weissflug, 1025 (Hel.)
Hötz, M. & Broscheit, A., 1018 (Hel.)
Hoxter, G. & Decoussau, D., (78) (Vms.)
Hsu, H. F., 255, 1008 (Hel.)
Hsu, S. C., Feng, L. P. & Chang, T. L., 952 (Mal.)
Huang, A. C. C., 1118 (Hel.)
—— & Wang, A., 145 (Hel.)
Huard, P., (1019) (Hel.)
——, Galliard, H. & Joyeux, B., (768) (Hel.)
—— & Nguyen-Xuân-Nguyen, (988) (Am.)
Huber, H., 466 (Am.)
Huebner, R. J., with Bell & Beck, 618 (Typh.)
——, with Jellison, Welsh & Elson, 731 (Typh.)
——, with Luoto, 730 (Typh.)
Huff, C. G., 1168 (Mal.)
Huggins, G., 1143 (B.R.)
Hughes, H. B. & Schmidt, L. H., 965 (Mal.)
Hughes, J. D., 748 (Am.)
Hughes, J. H., 162 (Ent.)
Hughes, L. E., with Jellison, Eneboe & Parker, 470 (R.F.)
Hughes, T. P., with Laemmert & Causey, 30 (Y.F.)
Huhtala, A., (140) (Hel.)
Hulshoff, A. A., 806 (Mal.)
——, Knoppers, A. T. & van Pinxteren, J. A. C., 175 (B.R.)
Humble, J. G., with Harland & Mann, 1015 (Hel.)
Humphreys, F. A., Campbell, A. G., Driver, M. W. & Hatton, G. N., 751 (R.F.)
Hunninen, A. V. & Young, M. D., 819 (Mal.)
Hunter, G. W., with Abbott, 379 (Hel.)
—— & Abbott, R. T., with Pan, C. & Stray, E., 379 (Hel.)
——, Bennett, H. J., Fry, N. H., See, J. & Greene, E., 380 (Hel.)
——, Dillahunt, J. A. & Dalton, H. C., 1007 (Hel.)
—— & Warren, V. G., 1112 (Hel.)
Hussey, K. L., 981 (Am.)
—— & Brown, H. W., 981 (Am.)
Hutchinson, M. P., with Harding, 824 (Tryp.)
Hutchison, J. H., 667 (Prot.)
Hutton, P. W., 526 (Tryp.), 1217 (Def. Dis.)
Huvé, P., (522) (Mal.)
Hwang, J. C., with Feng, Lin & Ting, 144 (Hel.)

I

Iaria, S. T., with Lacaz, Ferreira, Martins & Vega, 494 (Der.)
Ibarra, A. B., with Romero & Fallas, 371 (Lep.)
Ibrahim, K. G., (1117) (Hel.)
Ibrahim, M., Sorour, A. & Sherif, A., 40 (Am.)
d'Ignazio, C. & Mira, M. G., 1134 (Reports, etc.)
Ihle, J. E. W., with Swellengrebel, 675 (B.R.)
Imboden, C. A., Jr., with Coatney, Cooper, Culwell & White, 1177 (Mal.)
——, with Cooper & Coatney, 1057 (Mal.)
——, with ——, —— & Jeffery, 438 (Mal.)
Incho, H. H. & Deonier, C. C., 785 (Ent.)
Indian Research Fund Association, (730) *bis* (Typh.), 735 (Pl.), 814 (Mal.), 871 (Hel.), 1094 (Lep.)
Ingram, R. L., with Bohart, 1040 (Ent.)

Innes, J. R., 365, 366 *bis* (Lep.)
Internat. J. Leprosy, 370 (Lep.)
Ipinza, I., with Anselmo, Bahamondes, Lagos & Cortés, 1216 (Hel.)
Irigoyen Ramirez, A. I., with Diaz Marin, 854 (R.F.)
Iriondo, M., with Kouri & Antonio Peraza, 42 (Am.)
Irons, J. V., with Eads, Henderson & McGregor, 470 (R.F.)
Irving, J. T. & Selzer, G., 73 (Def. Dis.)
Isaza Mejia, G., 240 (Am.)
Iserle, J. & Maršálek, E., (406) (Prot.)
Israëls, M. C. G. & Sharp, J., (773) (Sp.)
Issa, D., with Robin, 205 (Mal.)
Issaly, A. S. & de Issaly, I. S. M., 355 (Pl.)
de Issaly, I. S. M., with Issaly, 355 (Pl.)
Issaris, P. C., with Livadas & Koroyannaki, 594 (Mal.)
Itano, H. A., with Pauling, Singer & Wells, (267) (Haem.)
Itoh, H., with Fukuzumi, Yamashina & Ohwada, 729 (Typh.)
——, Kumano, T., Masu, S. & Shimada, Y., 728 (Typh.)

J

Jachowski, L. A., Jr. & Stirewalt, M. A., 638 (Hel.)
Jackson, C. H. N., 17, 718 (Tryp.)
Jackson, E. B., with Crocker, Bennett, Snyder, Smadel, Gauld & Gordon, 617 (Typh.)
Jacobs, L., 1085 (Am.)
—— & Jones, F. E., 1229 (Prot.)
Jacobsen, A. K. F., 870 (Hel.)
Jacquemin, P., with Mandoul & Bernard, 896 (Ent.)
——, with Senevet & Mandoul, (276) (Ent.)
Jadin, J. & Fain, A., 431 (Mal.)
Jahan, P., with Canet, 261 (Hel.)
James, D. M., 161, (668) (Prot.)
——, with Kuitunen-Ekbaum, 877 (Hel.)
Janssen, E. & Le Roux, J. S., 1216 (Def. Dis.)
Janton, O. H., with Sigel, Scott & Henle, 1078 (Typh.)
Jarpa, A., Christen, R., Agosin, M. & Pizzi, T., 456 (Tryp.)
Jaswant Singh & Dalip Singh, 963 (Mal.)
—— & Kariapa, C. B., 963 (Mal.)
Jaujou, C., with Cavaillon, Adam, Bernard, Camain & Trinquier, 1179 (Mal.)
Javier Guillén, D., 1097 (Lep.)
Jawad, H. M., (407) (Prot.)
Jayawardene, M. D. S., 315 (Mal.)
Jeffery, G. M., with Cooper, Coatney & Imboden, 438 (Mal.)
——, with Eyles, 306 (Mal.)
Jelliffe, D. B., 83 (Der.), 495 (Misc. Dis.)
Jellison, W. L., Eneboe, P. L., Parker, R. R. & Hughes, L. E., 470 (R.F.)
——, Welsh, H. H., Elson, B. E. & Huebner, R. J., 731 (Typh.)
Jenkins, D. W., (458) (Typh.), 500, 1041 (Ent.)
—— & Hassett, C. C., 1041 (Ent.)
——, with ——, 498 (Ent.)
Jenkins, J. A., (57) (Hel.)
Jerace, F., 320 (Mal.)
Jiménez Díaz, C., Marina Fiol, C., Romeo Orbegozo, J. M. & Pérez Gòmez, A., 879 (Sp.)
Jirovec, O., 771 (Hel.)
Jochims, J. & Wrede, I., 258 (Hel.)
Johansen, F. A., Erickson, P. T. & Ross, H., 547 (Lep.)
——, ——, Wolcott, R. R., Meyer, W. H., Gray, H. H., Prejean, B. M. & Ross, H., 550 (Lep.)
Johnson, C. G., (668) (Ent.)
Johnson, C. M., with Elton, 732 (Y.F.)
Johnson, S. A., with Goldman, 552 (Hel.)
Johnson, T. H., Jr., with Young, 428 (Mal.)
Johnston, A. N., 210 (Mal.)
Johnstone, H. G. & Jones, O. W., Jr., 1107 (Hel.)
——, with Kessel, 43 (Am.)
Jonchère, H., 666 (Misc. Dis.)
—— & Martin, M., 659 (Der.)
Jones, B. M., 566 (Ent.)
Jones, B. S., 1056 (Mal.)
Jones, C. C., with Morton, (404) (Misc. Dis.)
Jones, E. A., with Smith, Cole & Clark, 279 (Ent.)
Jones, E. S., with Maegraith & Sculthorpe, 954 (Mal.)
Jones, F. E., with Jacobs, 1229 (Prot.)
Jones, H. A., with Travis, Morton & Robinson, (114) (Mal.)
Jones, J. I. M. & Wood, E. C., (165) (Lab.)
Jones, L. D., with Allen, 145 (Hel.)
Jones, M. F. & Newton, W. L., 624 (Am.)
——, with ——, 244, 468 (Am.)
Jones, O. W., Jr., with Johnstone, 1107 (Hel.)
Jones, R. W. & Holten, J. R., (415) (Ent.)
Jones, S. A., 1132 (Reports, etc.)
Jones, T. W. T., 322 (Mal.), 1233 (Ent.)
Jones, W. R., 748 (Am.)
de Jongh, D. K., with Winckel, Kok & Knoppers, 712 (Mal.)
Jope, H. M., with King, Gilchrist, Wootton, Donaldson, Sisson, Macfarlane, O'Brien, Peterson & Strangeways, 152 (Haem.)
Jordan, H., 1142 (B.R.)
Jordan, H. B., with Cantrell, 118 (Mal.)
Jordan, K., (1131) (Ent.)
Jose, F. R., with Salcedo, Bamba, Carrasco & Valenzuela, 560 (Def. Dis.)
J. National Malaria Soc., 898 (Reports, etc.)
J. Parasitology, 417 (Reports, etc.)
Joyeux, B., with Huard & Gailliard, (768) (Hel.)
Joyeux, C. & Baer, J. G., 555 (Hel.)
Joyner, L. P., with Fulton, 529 (Leish.)
——, with —— & Chandler, 1188 (Leish.)
Juneja, M. R., with Viswanathan & Ramachandra Rao, 1063 (Mal.)
Junior, J. G., with Rachou, Ricciardi & Luz, 588 (Mal.)

K

Kabler, P., with Adams, Cooney & Adams, 1038 (Prot.)
Kahn, M., 882 (Der.)
Kahn, M. C. & Offenhauser, W., Jr., 194, 195 (Mal.)
——, with Torrey, 15 (Mal.)
Kaiser, M. & Puntigam, F., 842 (Rab.)
Kalies, W., with Schlieper, 556, 868 (Hel.)

Kaira, S. L., with Narain, 857 (R.F.)
—— & Rao, K. N. A., 833, 835 (Typh.)
——, with ——, 857 (R.F.)
—— & Speechly, C. G. J., 834 (Typh.)
Kamal Bey, A. M., 738 (Chl.)
Kanazawa, K., 32 (Rab.)
Kane, C. A. & Most, H., 1013 (Hel.)
——, with ——, Lavietes, Schroeder, Behm, Blum, Katzin & Hayman, 1009 (Hel.)
Kanjilal, G. C., (405) (Misc. Dis.)
Kaplan, D., with Koch, 403 (Heat Str.)
Kaplan, F., with Kessel, 44 (Am.)
Kapur, K. B. & Das Gupta, P. R., 592 (Mal.)
Karandikar, K. R. & Munshi, D. M., (566) (Ent.)
Kariapa, C. B., with Jaswant Singh, 963 (Mal.)
Kartman, L., 500 (Ent.)
—— & Balock, J. W., (565) (Ent.)
Katzin, B., with Most, Kane, Lavietes, Schroeder, Behm, Blum & Hayman, 1009 (Hel.)
Kaufmann, H., with Donzelot, 613 (Typh.)
——, with —— & Vernant, 971 (Typh.)
Kaul, P. M., 353 (Pl.)
Kaul, S., 541 (R.F.)
Kayser, F. P., with Lehmann, 562 (Haem.)
Kee Hock Ooi, with Bruce-Chwatt, 1042 (Ent.)
Kelly, D. R., with Schnitzer, 826 (Tryp.)
Kelly, R. G. & Woodruff, C. E., 659 (Der.)
Kemény, T. T., with Veghelyi, Pozsonyi & Sós, 772 (Def. Dis.)
Kemp, G., 1227 (Prot.)
Kempski, H., 853 (Am.)
Kendall, S. B., 381 (Hel.)
Kenney, M., with Hewitt, Chan & Mohamed, 871 (Hel.)
Kenny, M., 592 (Mal.)
Kerharo, J. & Bouquet, A., 1102 (Lep.)
Kermack, W. O. & Storey, N. E., (522) (Mal.)
Kernevez, H. & Chassain, J., 825 (Tryp.)
Kershaw, W. E., 557 (Hel.)
——, with Gordon, 1214 (Hel.)
——, with ——, Crewe & Oldroyd, 1214 (Hel.)
Kessel, J. F., 981 (Am.)
—— & Johnstone, H. G., 43 (Am.)
—— & Kaplan, F., 44 (Am.)
——, Yeaman, A. & Holtzwart, F., 82 (Der.)
Kettle, D. S., 894 (Ent.)
Keys, A., with Henschel & Taylor, 519 (Mal.)
——, with Simonson, 519 (Mal.)
——, Wells, S., Hoffbauer, F. W., Taylor, H. L. & Henschel, A., 519 (Mal.)
Khalil Bey, M., 637 (Hel.)
Kho Lien Keng, 330 (Bl.)
Kikuth, W. & Bock, M., 227 (Typh.)
—— & Gönnert, R., 54, (637) (Hel.)
——, with Mudrow-Reichenow, 107 (Mal.)
King, E. J., Gilchrist, M., Wootton, I. D. P., Donaldson, R., Sisson, R. B., Macfarlane, R., Jope, H. M., O'Brien, J. R. P., Peterson, J. M. & Strangeways, D. H., 152 (Haem.)
King, W. V., with Smith, 670 (Ent.)
Kinkel, H., 107 (Mal.)
Kinsman, J. M., with Fishman, 203 (Mal.)
Kirk, R., 725 (Leish.)
——, Haseeb, M. A. & Davis, A. T., 1193 (Rab.)
—— & Lewis, D. J., 277, (499) (895) (Ent.)
Kirsch, E., with Chemnitz, 337 (Leish.)
Kirtisinghe, C., with Bibile, Cullumbine, Watson & Wikramanayake, (87) (Misc. Dis.)
——, with ——, ——, Wickramanayake & Watson, (77) (Haem.)
Kirwan, E. O'G., 1214 (Hel.)
Kissling, R. E., with von Brand, Tobie & Adams, 218 (Tryp.)
Kitaoka, M., with Berge & Gauld, 349 (Typh.)
Kitchen, S. F., with Smithburn, Goodner, Dick & Ross, 229 (Y.F.)
——, with ——, Mahaffy, Haddow & Smith, (620) (Den.)
Klimt, C., 34 (Rab.)
Klinger, W., 315 (Mal.)
Knight, A. A., Tarun, D. W. & Miller, J., 360 (Am.)
Knight, V., Ruiz-Sanchez, F., Ruiz-Sanchez, A. & McDermott, W., 458 (Typh.)
Knipling, E. F., with Madden, Lindquist & Longcoy, 278 (Ent.)
Knoppers, A. T., with Hulshoff & van Pinxteren, 175 (B.R.)
——, with Winckel, Kok & de Jongh, 712 (Mal.)
Knowles, F. A. & Terry, E. D., 858 (R.F.)
Knütgen, H., 199 (Mal.)
Knüttgen, H., with Ehlers, 638 (Hel.)
Koch, with Le Rouzic, 444 (Tryp.)
Koch, W. & Kaplan, D., 403 (Heat Str.)
Koenig, K., 713 (Mal.)
Koenigsfeld, E. G. H., 154 (Haem.)
Kohler, C. E., with Fox, 783 (Ent.)
Kohls, G. M., (1131) (Ent.)
——, with Brown, (897) (Ent.)
Köivastik, T., 398 (Vms.)
Kok, K., with Winckel, de Jongh & Knoppers, 712 (Mal.)
Koletsky, S., with Bueding, 863 (Hel.)
Koo, S. & Woo, D., 144 (Hel.)
Koprowski, H. & Black, J., 846 (Rab.)
——, Cox, H. R., Miller, M. S. & Florio, L., 1080 (Den.)
—— & LeBell, I., 979 (Rab.)
Korones, S. B., with Carter, (626) (Am.)
Koroyannaki, F. K., with Livadas & Issaris, 594 (Mal.)
Koshi, T., with Ranganathan & Sitaraman, 415 (Ent.)
Koshland, M. E. & Burrows, W., 1083 (Chl.)
Kostrzewski, J., 841 (Typh.)
Koszewski, B. J., with Moeschlin, 1191 (Typh.)
Kotcher, E., 981 (Am.)
Kothari, G. J., with Bhansali, 614 (Typh.)
Kotter, G. F., with Bras, Rijkebüsch & Djoa Liang Ham, 83 (Der.)
Kouo, C. C., with Lieou, 1193 *bis*, 1195 (Rab.)
Kourí, J., with Kourí, (1107) (Hel.)
Kourí, P., Iriondo, M. & Antonio Peraza, J., 42 (Am.)
—— & Kourí, J., (1107) (Hel.)
—— & Kouri Barreto, J., 767 (Hel.)
Kouri Barreto, J., with Kouri, 767 (Hel.)
Kouwenaar, W., 63 (Hel.)
Kozar, Z., 69, 250, 258 (Hel.)
Krakowsky, J. & Edelstein, E., (469) (R.F.)
Krause, G., with Fischer, 339 (Leish.)
Krey, W., 802 (Mal.)
Krishnan, K. S., with Raghavan, 1021, 1110 (Hel.)
——, with Ramakrishnan & Ramakrishna, 441 (Mal.)

Krishnan, K. V., 730 (Typh.)
Krouse, H., with McIntyre, 536 (Rab.)
Krumbiegel, I., 1080 (Pl.)
Krupp, I. M., with Sadun, Carrera & Allain, 852 (Am.)
Kruse, C. W., Hess, A. D. & Ludvik, G. F., 672 (Ent.)
Kryński, S., 728 (Typh.)
—— & Czuczwar, S., 126 (Typh.)
—— & Woyciechowska, S., 1074 (Typh.)
Kudicke, R., 346 (Typh.)
Kuhns, W. J., 154 (Haem.)
Kuitunen-Ekbaum, E., with Brown, Cronk, deSinner, Green & Gibbons, 558 (Hel.)
—— & James, D. M., 877 (Hel.)
Kumano, T., with Itoh, Masu & Shimada, 728 (Typh.)
Kumm, H. W., with Causey & Laemmert, 977 (Y.F.)
Kunstadter, R. H., Whitcomb, F. C. & Milzer, A., (83) (Der.)
Kurtpinar, H., with Furman, 1123 (Vms.)
Kuwata, T., Berge, T. O. & Philip, C. B, 531 (Typh.)

L

Labail, G., 744 (Am.)
LaBoccetta, A. C., with Dawson & Sherman, 566 (Ent.)
Laborde, R., with Montestruc & Blache, 1113 (Hel.)
LaCasse, W. J., 889 (Ent.)
Lacaz, C. da S., 1126 (Der.)
——, with de Almeida, 778 (Der.)
——, with ——, Ribeiro & de Azevedo, 775 (Der.)
——, laria, S. T., Ferreira, M., Martins, A. A. & Vega, V. S., 494 (Der.)
Lacerda, M. T., with Fonseca, Nunes & Pinto, 532 (Typh.)
——, with ——, Pinto & de Azevedo, 532 (Typh.)
——, with ——, ——, —— & Amaro, 532 (Typh.)
——, with ——, ——, Gander & de Azevedo, 532 (Typh.)
——, with ——, ——, Oliveira & da Gama, 532 (Typh.)
Lachmajer, J., 951 (Mal.)
Lackman, D. B., Parker, R. R. & Gerloff, R. K., 224 (Typh.)
——, with ——, de Prada & Bell, 460 (Typh.)
Lacroix, A., with Gelsi, (958) (Mal.)
Ladell, W. S. S., 157 (Heat Str.)
Ladkin, R. G., 526 (Tryp.)
Laemmert, H. W., Hughes, T. P. & Causey, O. R., 30 (Y.F.)
Laemmert, H. W., Jr., with Causey & Kumm, 977 (Y.F.)
Lagos, R., with Anselmo, Bahamondes, Ipinza & Cortés, 1216 (Hel.)
Lagrange, E. & Scheecqmans, G., 1105 (Hel.)
——, —— & Promel, R., 633 (Hel.)
Laguzzi, S. F., with Alvarado & Coll, 716 (Mal.)
Lahiri, B. N., with Brooks, d'Silva & Ahuja, (842) (Rab.)
Lahiri, M. N., with Singh, Malik & Ahuja, 848 (Chl.)
Laird, M., 432 (Mal.)
Laird, R. L., Drinnon, V. P. & Davis, A. B., 743 (Am.)
——, with Dubin & Drinnon, 327 (Mal.)
——, with McVay & Sprunt, 746 (Am.)
Lal, S. B., 496 (Misc. Dis.)
Lalouel, J., 264 (Def. Dis.), 823 (Tryp.), (975) (Typh.)
Lambault, E., with Deschiens & Bablet, 481 (Hel.)
Lambillon, J., 665 (Misc. Dis.)
—— & Beheyt, P., 239 (Am.)
Lampe, P. H. J. & Boenjamin, R., 543 (Lep.)
Lamy, with Siguier & Trélat, 247 (Ys.)
Lamy, L. & Bonnel, D., 165 (Lab.)
Lan, Y. & Lin, C., (1106) (Hel.)
Lancepleine, J., 755 (Lep.)
Lancet, 475 (Lep.)
Lang, K., Siebert, G. & Lönnecke, W., (478) (Hel.)
Languillon, J., with Blanc, 6 (Mal.)
——, with Mauzé, 1112 (Hel.)
Lanzo, A., 767 (Hel.)
Lapeyssonnie, L., with Gasq, 821 (Tryp.)
——, with Monnet, 1187 *ter* (Tryp.)
Lapierre, J., with Galliard, 1182, 1183 (Mal.)
Lara, C. B., 544 (Lep.)
——, with Nolasco, 546 (Lep.)
Laranja, F. S., Dias, E. & Nobrega, G., 22 (Tryp.)
Larsh, J. E., Jr., (482) (Hel.)
Laskowski, L., Pinkerton, H. & Greiff, D., (1192) (Bart.)
Lastra, 1093 (Lep.)
Lastra, I. & Coatney, G. R., 1068 (Mal.)
Laufer, M. W., with Denhoff, 150 (Hel.)
Laugier, G. V., 979 (Rab.)
Launoy, L., 17, 20, 21, 334 (Tryp.)
Lauret, 756 (Lep.)
——, with Laviron, 758 (Lep.)
Lauret, L., with Laviron & Schneider, 549 (Lep.)
Lavier, G., 510 (B.R.)
Lavietes, P. H., with Most, Kane, Schroeder, Behm, Blum, Katzin & Hayman, 1009 (Hel.)
Laviron & Lauret, 758 (Lep.)
Laviron, P., Lauret, L. & Schneider, J., 549 (Lep.)
van Lawick van Pabst, M. H., 199 (Mal.)
Lawler, A. L., with Norman, 400 (Der.)
Lawlor, W. K., 813 (Mal.)
Lawrence, R. F., (500) (Ent.)
LeBell, I., DeBoer, C. J., Hazz, E. K. & Cox, H. R., 845 (Rab.)
——, with Koprowski, 979 (Rab.)
Lebied, B., with Wanson & Courtois, 647 (Hel.)
——, with —— & Peel, 585 (Mal.)
LeBourdellès, B., 611 (Leish.)
Lecks, H. I., 88 (Misc. Dis.)
Lédo, J. F., with Deane, Freire, Cotrim, Sutter & de Andrade, 1065 (Mal.)
Lee, J. G., 779 (Misc. Dis.)
Leemann, A., with Mooser & Marti, 534 (Typh.)
Lees, K. A., with Cuthbertson, Lloyd & Emery, (77) (Haem.)
Lefranc, M., 342 *bis* (Leish.)
Le Gac, P. J. & Mullet, 998 (Ys.)
Legerton, C. W. & Chambers, W. L., 470 (R.F.)
Le Gouas, J., 1061 (Mal.)
Lehmann, H., 269, 488, 562 (Haem.)
—— & Baird, R. B., 76 (Haem.)
—— & Kayser, F. P., 562 (Haem.)
—— & Milne, A. H., 268 (Haem.)
—— & Raper, A. B., 267 (Haem.)

Leick, G., 485 (Hel.)
Lemenager, with Siguier, Piette, Coblentz & Trélat, 1173 (Mal.)
Lennette, E. H., with Meiklejohn, 1192 (Typh.)
——, Meiklejohn, G. & Thelen, H. M., 30 (Typh.)
Lent, H., (721) (Tryp.)
Lentze, F., 479 (Hel.)
Lentze, F. A., 865 (Hel.)
Leong, P. C., with Williamson, 560 (Def. Dis.)
Leprosy in India, 50, 364 (Lep.)
Leroux, G., with Hechenroth, Bécuwe & Mayan, 1114 (Hel.)
Le Roux, J. S., with Janssen, 1216 (Def. Dis.)
Le Rouzic, 446, 448, 449, 451 (Tryp.)
—— & Koch, 444 (Tryp.)
Lester, H. M. O., 819 (Tryp.)
Leusch, 993 (Am.)
Levaditi, C., Vaisman, A. & Barrat, L., (632) (Lep.)
——, ——, Chaigneau, H. & Barrat, L., 1103 (Lep.)
Levi Castillo, R., 306 (Mal.)
Levin, W. C., with Schneider & Haggard, 77 (Haem.)
Lewert, R. M., 817, 818 (Mal.)
Lewis, D. J., (300) (Mal.)
——, with Kirk, 277, (499), (895) (Ent.)
Lewis, E. A., 527 (Tryp.)
Lewthwaite, R., 837 (Typh.)
——, with Smadel, Woodward & Ley, 129 (Typh.)
Ley, H. L., Jr., with Smadel, Woodward & Lewthwaite, 129 (Typh.)
——, Woodward, T. E. & Smadel, J. E., 836 (Typh.)
Leyva, R. D., with Colorado Iris, 164 (Ent.)
Li, L., with Mao & Wu, 478 (Hel.)
Liang, K. C., with Watson & Paul, 806 (Mal.)
Liebermeister, K. & Zehender, E., 730 (Typh.)
Lie Kian Joe, 390 (Hel.)
—— & Bras, G., 861 (Hel.)
Lieou, Y. C., with Fournier, (980) (Chl.)
—— & Kouo, C. C., 1193 *bis*, 1195 (Rab.)
Lima, A. B., with Rachou & Lima, 805 (Mal.)
Lima, A. O. & Guimarães, N., (659) (Der.)
——, with Marsiaj, Guimarães & Cunha, 399 (Der.)
Lima, M. M., with Rachou & Lima, 805 (Mal.)
Limbos, P., 626 (Am.)
Lin, C., with Feng, Ting & Hwang, 144 (Hel.)
——, with Lan, (1106) (Hel.)
Lincicome, D. R., Thiede, W. H. & Carpenter, E., 987 (Am.)
Lindberg, K., 106 (Mal.), 1118 (Hel.)
Lindquist, A. W., with Deonier, Sullivan & Nottingham, 13 (Mal.)
——, with Madden, Longcoy & Knipling, 278 (Ent.)
——, Roth, A. R. & Yates, W. W., 892 (Ent.)
——, with Travis, 892 (Ent.)
Lindquist, W. D., (1211) (Hel.)
Ling, C. C., Cheng, W. J. & Chung, H. L., 380 (Hel.)
—— & Taur, S. S., 381 (Hel.)
Link, V. B., 847 (Pl.)
Linnell, W. H. & Sharp, L. K., (522) (Mal.)
Lints, H. A., Coatney, G. R., Cooper, W. C., Culwell, W. B., White, W. C. & Eyles, D. E., 1059 (Mal.)
Lints, H. A., with Coatney, Cooper, White, Culwell & Eyles, 1060 (Mal.)
Lippi, M., 766, 1017 (Hel.)
—— & Argiolas, N., 1218 (Haem.)
Lipton, M. M. & Freund, J., 845 (Rab.)
Liu, C. & Bang, F. B., 1105, 1106 (Hel.)
——, with Niu, 135 (Am.)
Livadas, G., 812 (Mal.)
Livadas, G. A., Koroyannaki, F. K. & Issaris, P. C., 594 (Mal.)
Llombart, A., 1095 (Lep.)
Llorente, A., with de Prada & Gay, 533 (Typh.)
Lloyd, J. F., with Cuthbertson, Emery & Lees, (77) (Haem.)
Lo, J. P., with Tokura, Tsai & Chen, 604 (Mal.)
Lock, J. A., with Goodwin & Goss, 1072 (Tryp.)
——, with ——, —— & Walls, 1071 (Tryp.)
Lodens, J. G., with van Goor, 807 (Mal.)
——, with Swellengrebel, 211 (Mal.)
Lodowski, C. H., with Sulkin & Hartman, 888 (Prot.)
Logan, J. A., 810 (Mal.)
Longcoy, O. M., with Madden, Lindquist & Knipling, 278 (Ent.)
Lönnecke, W., with Lang & Siebert, (478) (Hel.)
Looney, J. M. & Stein, T., 492 (Der.)
Lopez, H., with Garcia Lopez, Milanes, Spies, Lopez Toca & Aramburu, 396 (Sp.)
López-Neyra, C. R., 1017 (Hel.)
Lopez-Sanabria, U., with Rodriguez-Molina, Acevedo, Torres, Ramirez-Rodriguez & Diaz-Calderin, 1206 (Hel.)
Lopez Toca, R., with Garcia Lopez, Milanes, Spies, Aramburu & Lopez, 396 (Sp.)
——, with Milanés & Hernández Beguerie, 487 (Sp.)
Lotmar, R., with Wiesmann, 278 (Ent.)
Lotte, with Schneider, Marchalant, Buisson & Diallo, 449 (Tryp.)
Lou, T. C., with Fairbairn, (522) (Mal.)
Lourenço Marques, Moçambique, 217 (Tryp.)
Lourie, E. M., with Bülbring & Pardoe, 119 (Tryp.)
——, with Rollo & Williamson, 333 (Tryp.)
Lowe, J., 475, 1000 (Lep.)
—— & Smith, M., 474 (Lep.)
Lozano Morales, A., 812 (Mal.)
Lubinsky, G., 245 (Am.)
Lubitz, J. M., 307 (Mal.)
Lubran, M., 388, 1020 (Hel.)
Lucey, J. F., 1123 (Vms.)
Ludvik, G. F., with Kruse & Hess, 672 (Ent.)
Luis Carri, E., 42 (Am.)
Luis González, C., Potenza, L., Volgelsang, E., Anduze, P. J. & Torres, E. P., 645 (Hel.)
Lumsden, W. H. R., with Smithburn & Haddow, (32) (Den.)
Lund, H. Z., with Mercer, Bloomfield & Caldwell, 1109 (Hel.)
Luoto, L. & Huebner, R. J., 730 (Typh.)
Luttermoser, G. W., with Haskins, 1206 (Hel.)
——, Haskins, W. T. & Brady, F. J., 254 (Hel.)
Luz, E., with Rachou, Ricciardi & Junior, 588 (Mal.)
Lyon, E., 1220 (Der.)

M

Mac Arthur, W. P., 419, 899 (Reports, etc.)
McAuliff, J. P., with McChesney, 437 (Mal.)
McBride, O. C., Sullivan, W. N. & Fulton, R. A., 786 (Ent.)
MacCallum, F. O., Marmion, B. P. & Stoker, M. G. P., 226 (Typh.)
Maccarrone, A., with Buonomini, 439 (Mal.)
McCarten, W. G., with Griffin, 852, 1087 *bis* (Am.)
McChesney, E. W. & McAuliff, J. P., 437 (Mal.)
McConnachie, E. W., with Bishop, 525 *ter*. (Mal.)
McCoubrey, A., (1072) (Tryp.)
McDermott, W., with Knight, Ruiz-Sanchez & Ruiz-Sanchez, 458 (Typh.)
Macdonald, A. B., 51 (Lep.)
Macdonald, G., 4, 707, (Mal.) 787 (Reports, etc.), 1214 (Hel.)
McDonald, J. M., with O'Connor, 976 (Typh.)
Macfarlane, R. G., with King, Gilchrist, Wootton, Donaldson, Sisson, Jope, O'Brien, Peterson & Strangeways, 152 (Haem.)
Macfarlane, W. V., 765, 766 (Hel.)
McGhee, R. B., 14, 215, 1184 (Mal.)
—— & Trager, W., 1067 (Mal.)
——, with ——, 816 (Mal.)
McGovran, E. R., with Casanges & Chiles, (432) (Mal.)
MacGregor, R. B , 301 (Mal.)
McGregor, T., with Eads, Henderson & Irons, 470 (R.F.)
——, with Sullivan, Eads & Davis, 455 (Tryp.)
McIntyre, H. D. & Krouse, H., 536 (Rab.)
McKee, R. W., with Geiman, 1036 (Prot.)
McKenna, J., with Haworth & Whitfield, (360) (Am.)
Mackerras, I. M., 791 (Reports, etc.)
—— & Mackerras, M. J., 499 (Ent.)
Mackerras, M. J., with Mackerras, 499 (Ent.)
Mackichan, I. W., 526 (Tryp.)
Mackie, T. T., 981 *bis* (Am.)
—— & Sonnenberg, B., 788 (Reports, etc.)
——, Tuttle, R. L. & Simpson, T. W., 740 (Am.)
Mackinnon, J. E., 494 (Der.)
——, Ferrada-Urzúa, L. V. & Montemayor, L., 490 (Der.)
——, with García-Zorron, N., 1125 (Der.)
——, Montemayor, L. & Vinelli, H., 84 (Der.)
—— & Vinelli, H., 84, 882 (Der.)
Maclean, G., 673 (Reports, etc.)
McLetchie, J. L., 330 (Tryp.)
McMahon, J. J., with Covey & Myers, 71 (Hel.)
McMullen, D. B., 262 (Hel.)
McNair, P. K. & Others, 502 (Reports, etc.)
Macready, S. D., (599) (Mal.)
McRobert, G., 1214 (Hel.)
McVay, L. V., Jr., Laird, R. L. & Sprunt, D. H., 746 (Am.)
Madden, A. H., Lindquist, A. W., Longcoy, O. M. & Knipling, E. F., 278 (Ent.)
Maegraith, B. G., 284 (Reports, etc.)
——, Andrews, W. H. H. & Wenyon, C. E. M., 197 (Mal.)
——, with —— & ——, 198 (Mal.)
——, Jones, E. S. & Sculthorpe, H. H., 954 (Mal.)
de Magalhães, O., Freire, S. A. & Rocha, A., 839 (Typh.)
—— & Rocha, A., 346 (Typh.)
Magnusson, S. H., with Gard, Wahlgren & Gille 1039 (Prot.)
Mahaffy, A. F., with Haddow, 230 (Y.F.)
——, with ——, Gillett & Highton, 1231 (Ent.)
——, with Smithburn, Haddow, Kitchen & Smith (620) (Den.)
Mahboob, S., with Hall & Turner, (1090) (Am.)
Maier, E. H., 222 (Typh.), 644 (Hel.), 112 (Haem.)
Maillot, L. & Grjebine, A., 668 (Ent.)
Mainzer, F., 395 (Def. Dis.)
Mäkinen-Forel, M., 839 (Typh.)
Makstenieks, O., with Verlinde, Beekman & Copper, 1228 (Prot.)
——, with Winsser, 781 *bis* (Prot.)
Malaria and Mosquito Control, 101 (B.R.)
Malaya, 301 (Mal.)
Maldonado, J. F., Acosta Matienzo, J. & Thillet C. J., 553 (Hel.)
——, with Hernández Morales, Pratt & Olive González, 764 (Hel.)
——, with ——, Santiago Stevenson & Olive Gonzalez, 477 (Hel.)
——, with ——, ——, Pérez Santiago & Olive González, 761 (Hel.)
——, Hernández Morales, F., Vélez Herrera, F & Thillet, C. J., 872 (Hel.)
—— & Vélez Herrera, F., 553 (Hel.)
Malhotra, S. L., 260, (387) (Hel.)
Malik, K. S., with Singh, Lahiri & Ahuja, 84 (Chl.)
Mallet, R., with Méchali & Rodier, 310 (Mal.)
Malosetti, H., with Arana Iñiguez, Talice & Sa Julian, 555 (Hel.)
Manalang, C., 546 (Lep.)
Mandelbaum, R. A., with Murray, Baehr Shwartzman, Rosenthal, Doane, Weiss, Cohe & Snyder, 729 (Typh.)
Manderson, W. G., 347 (Typh.)
Mandoul, R., Bernard, F. & Jacquemin, P., 89 (Ent.)
——, with Senevet & Jacquemin, (276) (Ent.)
Maniscalco, G., 714 (Mal.)
Mankikar, D. S., with Green, 658 (Der.)
Mann, P. G., with Harland & Humble, 1015 (Hel
Manson-Bahr, P., 4 (Mal.), 121 (Tryp.), 113 (B.R.)
Mantz, F. A., Jr., Dailey, H. R. & Grocott, R. G 406 (Prot.)
Manwaring, J. H., 156 (Der.)
Manwell, R. D., 328 (Mal.)
Mao, C. P., Li, L. & Wu, C. C., 478 (Hel.)
——, with Olivier, 141 (Hel.)
Mara, L., 948 (Mal.)
Marangos, G., (144) (Hel.)
Marchalant, with Schneider, Lotte, Buisson & Diallo, 449 (Tryp.)
Marco Orts, F., with Forteza Bover & Garrigue Orellana, 854 (R.F.)
Marcovecchio, E., 300 (Mal.)
Maren, T. H., with Otto, 1115 (Hel.)
Mariani, M., with d'Alessandro & Burgio, 83 (Leish.)
Marie-Suzanne, with Noel, 367, 754 (Lep.)
Marill, F. G. & Friess, J., 253 (Hel.)
Marina Fiol, C., with Jiménez Díaz, Rome Orbegozo & Pérez Gòmez, 879 (Sp.)
Markey, R. L., 876 (Hel.)
Markowski, S., 382 (Hel.)

Marks, J. H., (643) (Hel.)
Marmion, B. P., with MacCallum & Stoker, 226 (Typh.)
Maroc, 227 (Typh.), 361 (R.F.)
Marot, J. T., with Armstrong & Elsdon-Dew, 628 (Am.)
Marousek, A. A., with Becker & Brodine, 325, 603 (Mal.)
Maršálek, E., with Iserle, (406) (Prot.)
Marsden, A. T. H., (667) (Misc. Dis.)
Marsiaj, N., Guimarães, N., Cunha, J. P. & Lima, A. O., 399 (Der.)
Martelli, T. & Zaffino, C., 1118 (Hel.)
Marti, H. R., with Mooser & Leemann, 534 (Typh.)
Martin, L. A., with Blanc & Bruneau, 130 (Typh.)
Martin, M., with Jonchère, 659 (Der.)
Martin, R., Montel, L. R. & Vargues, R., 974 (Typh.)
Martinez, R., with Galliard & Brumpt, 888 (Misc. Dis.)
Martínez Báez, M., 149 (Hel.)
Martins, A. A., with Lacaz, Iaria, Ferreira & Vega, 494 (Der.)
Martins, A. V., 760 (Hel.)
Martins, C. M., with Rachou & Neto, 197 (Mal.)
Martins, F., 609 (Tryp.)
Marty, J., with Germain & André, 1113 (Hel.)
Mason, S. F., (437) (Mal.)
——, with Hammick, (437) *bis* (Mal.)
Masquelier, J. & Bailenger, J., 480 (Hel.)
Massias, C., 111 (Mal.)
Masu, S., with Itoh, Kumano & Shimada, 728 (Typh.)
Matheson, R., 576 (B.R.)
Mathis, C., 359 (Am.)
Mathis, W., Ferguson, F. F., Upholt, W. M. & Quarterman, K. D., 892 (Ent.)
—— & Quarterman, K. D., 442 (Mal.)
Matilla, V., 1044 (B.R.)
Matilla Gomez, V. & Piedrola Gil, G., 356 (Pl.)
Mattes, O., (381) (Hel.)
Mattingly, P. F., 304, 583 (Mal.)
Matumoto, M., 235 (Pl.)
Maujol, L., with Denoix & Moine, 100 (Reports, etc.)
Mauri, A. C., with Rheinboldt, Berti, Perego, Rieckmann, Rzeppa, Hadler & de Souza Lima, 548 (Lep.)
Maurice, A., with Gaud & Fauré, 803 (Mal.)
Maurin, J., with Barski, 843 (Rab.)
Mauritius, 321 (Mal.)
Mauzé, J., 1001 (Lep.)
—— & Languillon, J., 1112 (Hel.)
Mayan, L., with Heckenroth, Bécuwe & Leroux, 1114 (Hel.)
Mayer, H., 347 (Typh.)
Mayer, H. F., with Nájera, 1107 (Hel.)
Mayer, M., (607) (Tryp.), 1192 (Bart.)
——, with Briceño Rossi, 644 (Hel.)
——, Convit, J. & Pifano C. F., 611 (Leish.)
—— & Pifano C. F., 590 (Mal.), (607) (Tryp.), 762 (Hel.)
——, with ——, 609 (Tryp.)
——, with ——, Römer, Domínguez, Medina & Benaím Pinto, 723 (Tryp.)
——, di Prisco, J. & di Prisco, R., 634 (Hel.)
Mazza, S., (722) (Tryp.)
Mazzotti, L., 68, 148, 252 (Hel.), 750 (R.F.)
—— & Olarte, J., 657 (Der.)
—— & Osorio, M.T., 771 (Hel.)
Meaker, R. E., 774 (Ep. Dropsy)
Méchali, D., with Gaud & Clier, 595 (Mal.)
——, with —— & Schneider, 1176 (Mal.)
——, with Messerlin, 897, 898 (Ent.)
——, Rodier, J. & Mallet, R., 310 (Mal.)
——, with Schneider, 204 (Mal.)
Medina, G., 249 (Lep.)
Medina, R., with Miguel Itriago, 752 (Ys.)
——, with Pifano, Mayer, Römer, Domínguez & Benaím Pinto, 723 (Tryp.)
Megaw, J. W. D., 577 (B.R.)
Mehlman, B., with von Brand & Nolan, 56 (Hel.)
——, with Tobie & von Brand, 604 (Tryp.)
Meiklejohn, G. & Lennette, E. H., 1192 (Typh.)
——, with —— & Thelen, 30 (Typh.)
de Meillon, B., 1043 (Ent.)
Meleagros, J., (112) (Mal.)
Meleagros, J. M., 136 (Am.)
Meleney, H. E., with Yolles & Moore, 142 (Hel.)
Melgarejo de Talavera, S., with Ginés & Gould, 400 (Der.)
Melick, D. W., 402 (Der.)
de Mello, M. T., 271 (Der.)
Mendheim, H. & Scheid, G., 68, 69, 1002 (Hel.)
——, with —— & Amenda, 1208 (Hel.)
Meneghello, J., Espinoza, J. & Coronel, L., 72 (Def. Dis.)
Meneses Rubiánes, C. & Avendaño Escalante, S., 1216 (Hel.)
de Menezes, M., 336 (Tryp.)
Menk, W. & Mohr, W., 112 (Mal.), 238 (Am.)
Menon, P. S., Tulpule, P. G. & Patwardhan, V. N., 1120 (Def. Dis.)
Menu, P., with Ceccaldi, Trinquier & Arnoult, 824 (Tryp.)
Mer, G. G. & Davidovici, S., 567 (Ent.)
Mercado, T. I., (1131) (Ent.)
Mercer, E. M., (735) (Rab.)
Mercer, R. D., Lund, H. Z., Bloomfield, R. A. Caldwell, F. E., 1109 (Hel.)
Merle, F., 242 (Am.)
Messerlin, A. & Mechali, D., 897, 898 (Ent.)
Messinezy, D. A., 418 (Reports, etc.)
Meyer, K. F., 737 (Pl.)
Meyer, W. H., with Johansen, Erickson, Wolcott, Gray, Prejean & Ross, 550 (Lep.)
Michaelides, N. C., 365 (Lep.)
Michaelson, J. B., with Hallman & DeLamater, 1086 (Am.)
Michini, L. J., with Griffin, 1087 (Am.)
Mickal, A., with Beacham & Beacham, 881 (Haem.)
Micks, D. W., 117, 328 (Mal.)
Miguel Itriago, P. & Medina, R., 752 (Ys.)
Milanes, F., with Garcia Lopez, Spies, Lopez Toca, Aramburu & Lopez, 396 (Sp.)
——, Hernández Beguerie, R. & Lopez Toca, R., 487 (Sp.)
Mille, R., with Galliard & Robinson, 60, 146 (Hel.)
Miller, D. & Birsner, J. W., 401 (Der.)
Miller, J., with Knight & Tarun, 360 (Am.)
Miller, J. C., with Bovarnick, (970) (Typh.)
——, with —— & Snyder, 832 (Typh.)
Miller, J. W., with Most & Grossman, 1200 (Am.)
——, with ——, —— & Conan, 1089 (Am.)

Miller, L., with Cohen, 1031 (Der.)
Miller, M. S., with Florio & Mugrage, 978 *bis* (Den.)
——, with Koprowski, Cox & Florio, 1080 (Den.)
Milne, A. H., with Lehmann, 268 (Haem.)
Milzer, A., with Kunstadter & Whitcomb, (83) (Der.)
Minning, W. & Vogel, H., 863 (Hel.)
——, with ——, 635 (Hel.)
Mira, M. G., with d'Ignazio, 1134 (Reports, etc.)
Misra, B. G., with Raghavan, 1023 (Hel.)
Mitchell, J. P., 1142 (B.R.)
Mitra, A. K., with Mukherjee, 954 (Mal.)
Modern, F. W. S., with Brawley, 348 (Typh.)
Moeschlin, S. & Koszewski, B. J., 1191 (Typh.)
Moffatt, J. S., (958) (Mal.)
Mofidi, C., with Baltazard, Bahmanyar, Habibi & Seydian, 1091 (R.F.)
——, with ——, ——, Seydian & Habibi, 995 (R.F.)
Moggridge, J. Y., 332, 333, 606 (Tryp.)
Mohamed, H., with Hewitt, Kenney & Chan, 871 (Hel.)
Mohammed, A. H., 77 (Vms.)
Mohapatra, G. S., 316 (Mal.)
Mohiuddin, A., 828 (Leish.)
Mohr, W., 38 (Am.), 711 (Mal.)
——, with Beckermann, 38 (Am.)
——, with Menk, 112 (Mal.), 238 (Am.)
—— & Westphal, A., 1228 (Prot.)
Moine, M., with Denoix & Maujol, 100 (Reports, etc.)
Mole, R. H., with Horne, 80 (Der.)
Molesworth, B. D. & Narayanaswami, P. S., 474 (Lep.)
——, with Simpson, 860 (Lep.)
Monnet, A. & Lapeyssonnie, L., 1187 *ter* (Tryp.)
Monteiro, A. B., with Silva, (266) (Def. Dis.)
Monteiro, E. S., 147 (Hel.)
Montel, L. R., with Martin & Vargues, 974 (Typh.)
Montel, M. L. R., 39 (Am.)
de Montemayor, L., 271 (Der.)
Montemayor, L., with Artagaveytia-Allende, 493 (Der.)
——, with Mackinnon & Ferrada-Urzúa, 490 (Der.)
——, with —— & Vinelli, 84 (Der.)
Montestruc, E., Blache, R. & Laborde, R., 1113 (Hel.)
——, Caubet, P. & Blaché, R., 1094 (Lep.)
Montézin, G., with Schneider, 43 (Am.), 1066 (Mal.)
——, with —— & Decourt, 325 (Mal.)
Moore, D. V., with Yolles & Meleney, 142 (Hel.)
Moormann, H., 478 (Hel.)
Mooser, H., Marti, H. R. & Leemann, A., 534 (Typh.)
Moretti, G. F., with Bonnin, 1108, 1115 (Hel.)
——, with —— & Guignard, 742 *ter*. (Am.)
Morin, H. G. S., with Chabaud, (975) (Typh.)
Morin, M., with Fouquet, 841 (Typh.)
Morlan, H. B., with Emmons & Hill, 491 (Der.)
——, Hill, E. L. & Schubert, J. H., 458 (Typh.)
Moruzzi, T., 582 (Mal.)
Morris, K. R. S., 19, 335 (Tryp.)
Morris, R. M., 96 (Reports, etc.)
Morton, F. A., with Travis, Jones & Robinson, (114) (Mal.)
Morton, P. H. & Jones, C. C., (404) (Misc. Dis.)
Mosna, E. & Alessandrini, M., 1180 (Mal.)
Most, H., with Kane, 1013 (Hel.)
——, Kane, C. A., Lavietes, P. H., Schroede E. F., Behm, A., Blum, L., Katzin, B. Hayman, J. M., Jr., 1009 (Hel.)
——, Miller, J. W. & Grossman, E. J., 1200 (Am.
——, ——, Grossman, E. B. & Conan, N., Jr 1089 (Am.)
Motta, J., 176 (B.R.)
Motulsky, A. G., with Singer & Wile, (881 (Haem.)
Moulder, J. W., 118 (Mal.), 1034 (Prot.)
de Moura, A. M., 1099 (Lep.)
Moynihan, I. W. & Musfeldt, I. W., 71 (Hel.)
Mudrow, L. & Reichenow, E., 819 (Mal.)
Mudrow-Reichenow, L., (1060) (Mal.)
—— & Kikuth, W., 107 (Mal.)
Mugrage, E. R., with Florio & Miller, 978 *bi* (Den.)
Muir, E., 552 (Lep.)
—— & Roy, A. T., (1001) (Lep.)
Muirhead-Thomson, R. C., 439 (Mal.)
Mukerjee, with Dharmendra, 367 (Lep.)
Mukerji, B., with Chowdhuri, (50) (Lep.)
Mukherjee, N., with Dharmendra, 755 (Lep.)
Mukherjee, S. K., 41 (Am.), 772 (Def. Dis.)
—— & Mitra, A. K., 954 (Mal.)
Mukherji, M., with Das, (261) (Hel.)
Mulhern, T. D. & Hart, J. T., Jr., (409) (Ent.)
Müller, F. P., 499 (Ent.)
Muller, G. R., with Swartzwelder, 749 (Am.)
Müller, R. W., 641 (Hel.)
Mullet, with Le Gac, 998 (Ys.)
Muñoz Monteavaro, C., with Pla, 359 (Am.)
Munro, A. F., 884 (Heat Str.)
Munshi, D. M., with Karandikar, (566) (Ent.)
Mura, D., 1088 (Am.)
Murdock, J. R., 370 (Lep.)
Murgatroyd, F. & Woodruff, A. W., 262 (Hel.)
Murphy, F. D. & Pascall, K. G., 555 (Hel.)
Murphy, M. A., with Phillips, (1226) (Misc. Dis.)
Murray, E. S., Baehr, G., Shwartzman, G., Mandelbaum, R. A., Rosenthal, N., Doane, J. C., Weiss, L. B., Cohen, S. & Snyder, J. C., 729 (Typh.)
——, with Fuller & Snyder, 127 (Typh.)
Murray, R. E., (1123) (Vms.)
Murtadha, M., with Watson, Zahawi & Naji, 1203 (Hel.)
Musfeldt, I. W., with Moynihan, 71 (Hel.)
de Muynck, A., (266) (Haem.)
Myers, H. L., with Covey & McMahon, 71 (Hel.)

N

Nagendra, S. & Narayana Murphy, K. S., 962 (Mal.)
Nair, C. P., 440, 1064 (Mal.)
——, with Singh & Ray, 1181 (Mal.)
——, with ——, —— & Basu, 1186 (Mal.)
Nájera, L., 1004 (Hel.)
——, with Romaña, Conejos & Abalos, 125 (Leish.)
Nájera, L. E. & Mayer, H. F., 1107 (Hel.)
Naji, A. F., with Watson, Zahawi & Murtadha, 1203 (Hel.)
Nandi, A. K., 200 (Mal.)
Napier, L. E., 125, 341 (Leish.)
Narain, S. & Kalra, S. L., 857 (R.F.)

Narayana Murphy, K. S., with Nagendra, 962 (Mal.)
Narayanaswami, P. S., with Molesworth, 474 (Lep.)
Nascimbene, A., with Pratesi, Raffa & Babudieri, 1091 (R.F.)
Nash, T. A. M. & Davey, J. T., 718 (Tryp.)
Nauck, E. G., 5 (Mal.)
Navarranne, P., 642 (Hel.)
Néel, R. & Payet, M., 996 (R.F.)
——, —— & Gonnet, C., 137 (R.F.)
Neghme, A., (891) (Ent.)
——, Donckaster, R. & Silva, R., (1107) (Hel.)
——, with Pizzi, Agosin, Christen & Hoecker, 455 (Tryp.)
—— & Silva, R., 464, 1197 (Am.)
——, ——, Vilches, R. & Alfaro, E., 722 (Tryp.)
Negroni, P., (271) *bis* (Der.)
—— & Radice, J., 1124 (Der.)
Nelson, J. W., 460 (Typh.)
Netherlands Red Cross Feedingteam, 511 (B.R.)
Neto, J. A. F., with Rachou & Martins, 197 (Mal.)
Nettel F., R., 646 *bis* (Hel.)
Newbold, C. E. & Gillette, H. P. S., 809 (Mal.)
Newsome, J., 378, (476) (Hel.)
Newton, W. L., 981 (Am.)
—— & Jones, M. F., 244, 468 (Am.)
——, with ——, 624 (Am.)
Ngu, D. V., with Galliard, (565) (Ent.)
Nguyen-Dinh-Diep, with Bruneau, (975) (Typh.)
Nguyen-Xuân-Nguyen, with Huard, (988) (Am.)
Nicholls, L., 394 *bis* (Def. Dis.)
Nicholson, C. C., 559 (Def. Dis.)
Nicholson, H. P. & Vetter, M. H., 848 (Pl.)
Nicol, B. M., 151 (Def. Dis.)
Nicolle, M., 954 (Mal.)
Nigeria, 330 (Tryp.)
Nikolic, J. & Weiser, J., 1108 (Hel.)
Niño, F. L., 780 (Misc. Dis.)
Nishioka, K., with Tamiya, Hazato, Yamamoto & Shimojo, 344 (Typh.)
Niu, P. & Liu, S., 135 (Am.)
Nobrega, G., with Laranja & Dias, 22 (Tryp.)
Nodenot & Porte, 242 (Am.)
Noé C., J. & Silva C., R., (609) (Tryp.)
Noel, R. & Marie-Suzanne, 367, 754 (Lep.)
Noguès, C., with Sigalas & Pautrizel, 357 (Am.)
Nolan, M. O. & Berry, E. G., 143 (Hel.)
——, with von Brand & Mehlman, 56 (Hel.)
Nolasco, J. O. & Lara, C. B., 546 (Lep.)
Nor Eldin, G. & Baz, I. I., 383 (Hel.)
Norman, I. L. & Lawler, A. L., 400 (Der.)
Noronha Miranda, R., (773) (Def. Dis.)
Northcott, C. H., 279 (Reports, etc.)
Norton, H. E., with Goldenson, Novak & Sass, 164 (Ent.)
Norval, J., with Seiler, 1107 (Hel.)
Nory, J., with Ravina & Avril, 404 (Misc. Dis.)
Nottingham, E., with Deonier, Raun, Peek & Davis, 443 (Mal.)
——, with ——, Sullivan & Lindquist, 13 (Mal.)
Novak, C. F., with Goldenson, Norton & Sass, 164 (Ent.)
Novy, F. G., Jr., 80 (Der.)
Nunes, M. A., with Fonseca, Pinto & Lacerda, 532 (Typh.)
Nuñez Andrade, R., 545 (Lep.)
Nutt, A. B., Wright, T., Bowie, J. M. & Beattie, C. P., 89 (Prot.)
Nyka, W., 531 (Typh.)

O

Obermayer, M. E., Bonar, S. C. & Rosenquist, R., 1098 (Lep.)
O'Brien, J. R. P., with King, Gilchrist, Wootton, Donaldson, Sisson, Macfarlane, Jope, Peterson & Strangeways, 152 (Haem.)
de Ocampo, G., 661 (Oph.)
O'Connor, J. L. & McDonald, J. M., 976 (Typh.)
Odendaal, W. A., with Steyn, P. J., 73 (Def. Dis.)
O'Dwyer, J. J., 285 (Reports, etc.)
Oelrichs, L., with Bieling, 344, 345 *bis* (Typh.)
Offenhauser, W., Jr., with Kahn, 194, 195 (Mal.)
Ogaki, M., 16 (Mal.)
Ohwada, H., with Fukuzumi, Itoh & Yamashina, 729 (Typh.)
Okan, S., 595 (Mal.)
Olarte, J., with Mazzotti, 657 (Der.)
——, with Varela, 1220 (Der.)
Oldroyd, H., 1230 (Ent.)
——, with Gordon, Kershaw & Crewe, 1214 (Hel.)
Oliphant, J. W. & Tibbs, R. O., 732 (Den.)
de Oliveira, G. G., 258 (Hel.)
Oliveira, J., with Fonseca, Pinto, da Gama & Lacerda, 532 (Typh.)
de Oliveira Lima, S., with Genú, 551 (Lep.)
——, with —— & Santos, 551 (Lep.)
Oliver González, J., with Hernández Morales, Pratt & Maldonado, 764 (Hel.)
——, with ——, Santiago Stevenson & Maldonado, 477 (Hel.)
——, with ——, ——. Pérez Santiago & Maldonado, 761 (Hel.)
Olivier, L. & Mao, C. P., 141 (Hel.)
Omer-Cooper, J., 476 (Hel.)
Onabamiro, S. D., 390 (Hel.)
Openshaw, H. T., with Battersby, (360) (Am.)
Orduz-Duarte, A., with Renjifo-Salcedo, (1022) (Hel.)
Orr, J. L., with Graham, 763 (Hel.)
Ortega Canet, F., 53 (Hel.)
Ortiz O., P., with Calero, 651 (Hel.)
Ortiz Machado, O. & Beyra Alemany, M., (1057) (Mal.)
Ortiz Mariotte, C. & Varela, G., 225 (Typh.)
——, ——, Roch, E. & Gonzalez Robles, F., 973 (Typh.)
Osorio, M. T., with Mazzotti, 771 (Hel.)
——, with Quintanar, 263 (Hel.)
Osorno-Mesa, E., with Renjifo-Salcedo, (970) (Tryp.)
Otto, G. F., 981 (Am.)
—— & Maren, T. H., 1115 (Hel.)
——, with Velat & Weinstein, 625 (Am.)
Ouary, G., 240 (Am.)
Ovazza, M., 1022 (Hel.)
——, with Stefanopoulo, 390 (Hel.)
Overman, R. R., Bass, A. C., Davis, A. K. & Golden, A., 116 (Mal.)
——, with Golden, 110 (Mal.)
Oyama, V., with Germuth & Eagle, 1188, 1189 (Leish.)
van Oye, E., with Peel, 1111 (Hel.)

P

Packchanian, A., 720 (Tryp.)
Paes, F. J. A., 4 (Mal.)
Pagast, F., with Eichler, 431 (Mal.)
Page, A. B. P. & Blacklin, R. E., 93 (Ent.)
——, Swinger, A. & Blacklin, R. E., 93 (Ent.)
Pages, R., with Blanc & Bruneau, 663 (Oph.)
Pai, R., (161), (782) (Ent.)
Palmer, E. D., 640 (Hel.)
Palmquist, J., with Haussükin, Goodwin & Brown, 607 (Tryp.)
Pan, C., with Hunter, Abbott & Suay, 379 (Hel.)
Pan, H. S., with Giroud & Ciaccio, 913 (Typh.)
P'An, H. S., Tchan, Y. T. & Pochon, J., 847 (Pl.)
Panama Canal, 99 (Reports, etc.)
Panganiban, E. C., with Salcedo, Pedroche & DeLeon, 73 (Def. Dis.)
Pani, A., 862 (Hel.)
Panisset, M., with Guy & Frappier, 82 (Der.)
Panja, D., with Ghosh & Dey, (1221) (Der.)
Panja, G. & Gupta, S. K., 555 (Pl.)
Pannier, L., with Wanson & Borgers, 873 (Hel.)
Pansini, R. & Pinassi, C., (1017) (Hel.)
Panthier, R., 190 (Mal.)
Pantani, R., with Pieri, 200 (Mal.)
Papayannis, T., 70 (Hel.)
Papoiotti, J., 722 (Tryp.)
Paruense, W. L., (90) (Prot.), (517) (Mal.)
Paramonow, S. J., (409) (Ent.)
Parande, A. S., (1208) (Hel.)
Pardo Castelló, V. & Piñeyro, R., 370 (Lep.)
Pardoe, U., with Bülbring & Lourie, 119 (Tryp.)
Paredes-Manrique, R., with Hernandez-de-Paredes, 724 (Tryp.)
Parent, J., 656 (Haem.)
Parker, A. H., 406 (Ent.)
Parker, J. P., Ewing, P. L. & Emerson, G. A., 969 (Tryp.)
Parker, R. R., with Jellison, Eneboe & Hughes, 470 (R.F.)
——, with Lackman & Gerloff, 224 (Typh.)
——, de Prado, J., Bell, E. J. & Lackman, D. B., 446 (Typh.)
—— & Sussman, C., 130 (Typh.)
Parkes, A. S., with Bruce, (165) (Lab.)
——, with Short, (1165) (Lab.)
Parrot, G., with Parrot, 344 (Typh.)
Parrot, L., (277) (Ent.)
—— & Canamet, A., 423, (905), 1168 (Mal.)
—— & Parrot, G., 344 (Typh.)
Pascall, K. G., with Mumby, 555 (Hel.)
Pashar, N. A., 726 (Leish.)
Patnaik, T., 115 (Mal.)
Patwardhan, V. N., with Menon & Tulpule, 1120 (Def. Dis.)
——, with Ranganathan, 266 (Haem.)
Paul, J. H., with Watson & Liang, 806 (Mal.)
de Paula, A., 628 (Am.)
Pauling, L., Itano, H. A., Singer, S. J. & Wells, I. C., (267) (Haem.)
Paumgard, R., Beman, A. & Baslanger, J., 375 (Hel.)
——, with Sigalas & Noguès, 357 (Am.)
Pavlovsky, E. N., 1079 (Den.)
Payet, M., with Neel, 996 (R.F.)
——, with —— & Gonnet, 187 (R.F.)
Payne, S., 129, (129) (Typh.)
Peake, J. D. & Eskridge, M., 745 (Am.)
Pearse, A. S., 1138 (Reports, etc.)
Pearson, A. W., with Wright, 563 (Haem.)
Pearson, G. H., 662 (Oph.)
Pécher, Y., with Ravina & Avril, 855 (R.F.)
——, with ——, Guerin & Clavel, (989) (Am.)
Pedroche, A., with Salcedo, Panganiban, DeLeon, 73 (Def. Dis.)
Peak, E. H., with Deornier, Raun, Davis & Nottingham, 443 (Mal.)
Peel, E., with Chardome, 65 (Hel.)
——, with Henrard, 66 (Hel.)
—— & van Oye, E., 1111 (Hel.)
——, with Wanson, 65 (Hel.)
——, with —— & Lebied, 585 (Mal.)
Peeters, E. M. E., 204 (Mal.)
Pelly, R. L., with Sundararaman, 323 (Mal.)
Pelaez, D., with Soberon y Parra, 360 (Am.)
Pellegrino, J., 125 (Tryp.)
—— & Borrotchin, M., 23 (Tryp.)
Pellissier, A., with Ceccaldi & Arnoult, 18, 5 (Tryp.)
——, with ——, Trinquier & Arnoult, 528 (Tryp.)
——, Trequereau, P. & Trinquier, E., 617 (Typ
—— & Trinquier, E., 459 (Typh.)
Pelous, Y., with Sohier, 1090 (Am.)
Pelloux, A., with Decourt & Dupoux, 140 (He
Penido, H. M., with Pinto, 765 (Hel.)
——, Pinto, D. B. & de Souza, E. F., 600 (Ma
Penn, G. H. & Coleman, S. A., (306) (Mal.)
Pelluaux Rose, A., 365 (Hel.)
Péquignot, H., with Worms & Held, 971 (Typ
Pereda Regnera, A., 426, 439 (Mal.)
Perego, C., with Berti, Rieckmann & Rzepp 372 (Lep.)
——, with Rheinboldt, Berti, Rieckmann, Rzepp Mauri, Hadler & de Souza Lima, 548 (Lep.)
Pereira, H. G. & Travassos, J., 1076 (Typh.)
——, with —— & Vasconcelos, 346 (Typh.)
——, with Vasconcelos & Travassos, 1075 (Typ
Pereira Barreto, M. & Pessôa, S. B., 782 (Ent.)
Peren, E., with Suarez, Spies, Hernandez-Moral & Benitez-Gautier, 397 (Sp.)
Perez, R., with Beltrán, 609 (Tryp.)
Pérez Ara, A., 1213 (Hel.)
Perez Gallardo, F., Clavero, G. & Hernández Fernández, S., 840 (Typh.)
——, with Clavero Del Campo, 222 (Typh.)
Perez Gómez, A., with Jimenez Diaz, Marina Fi & Romeo Ortegozo, 879 (Sp.)
Perez Santiago, E. & Hernández Morales, F., 119 (Am.)
——, with ——, Santiago Stevenson, Olive González & Maldonado, 761 (Hel.)
Perry, E. L., 707 (Mal.)
Perry, W. J., 259 (Hel.), 497, 890 (Ent.)
—— & Bodenlos, L. J., (786) (Ent.)
Peru, 964 (Mal.)
Pessôa, S. B. & Andrade, Z. de A., 1111 (Hel.)
——, with Pereira Barreto, 782 (Ent.)
—— & Silvany, A., Jr., 1085 (Am.)
Peters, L., Welch, A. D. & Higashi, A., 147 (Hel.)
Peters, W., 1027 (Def. Dis.)
Peterson, E. T., with Deutsch, 840 (Typh.)
Peterson, J. M., with King, Gilchrist, Wootten, Donaldson, Sisson, Macfarlane, Jopé, O'Brien & Strangeways, 152 (Haem.)
Peus, F., 1231 (Ent.)
Pfeiffer, K. H., (405) (Misc. Dis.)

Pfeiffer, D. H., 531 (Typh.)
Pfister, R., 630, 699 (R.F.)
Pham-Khac-Quang, with Rivoalen, (975) (Typh.), (988) (Am.)
Pham-Ngoc-Thach, (1017) (Hel.)
Phiem, N. H., with Galliard & Grenierboley, (1040) (Ent.)
Philip, C. B., with Kuwata & Berge, 531 (Typh.)
—— & Traub, R., 566 (Ent.)
Phillips, B. P., 624 (Am.)
—— & Rees, C. W., 741 (Am.)
Phillips, P. & Murphy, M. A., (1226) (Misc. Dis.)
Pick, F., 134 (Am.)
——, with Deschiens, 16 (Mal.)
Pickett, A. D., 501 (Ent.)
Piedrola Gil, G., 1044 (B.R.)
——, with Matilla Gomez, 356 (Pl.)
Piekarski, G., 21 (Tryp.), 462 (Am.), 1129 (Prot.)
Pieraerts, G., 265, 653, 1121 (Def. Dis.)
Pieri, J. & Panzani, R., 200 (Mal.)
Piers, F., (660) (Der.)
Piers, F. O., 203 (Mal.)
Piette, with Siguier, Coblentz, Lemenager & Trélat, 1173 (Mal.)
Pifano C., F., 727 (Leish.), 762 (Hel.)
—— & Mayer, M., 609 (Tryp.)
——, with ——, 590 (Mal.), (607) (Tryp.), 762 (Hel.)
——, with —— & Convit, 611 (Leish.)
——, with ——, Römer, M., Domínguez, E., Medina, R. & Benaim Pinto, E., 723 (Tryp.)
—— & Römer, M., (656) *bis* (Vms.)
Pille, G., 885 (Heat Str.), (898) (Ent.)
——, with Pluchon, 468 (Am.)
Pinakapani, S., with Raman & Ramamurthy, 643 (Hel.)
Piney, A. & Hamilton-Paterson, J. L., 173 (B.R.)
Piñeyro, R., with Pardo Castelló, 370 (Lep.)
Pinkerton, H., with Laskowski & Greiff, (1192) (Bart.)
Pinotti, M., 116, 212, 1064, (1065) *bis* (Mal.), (1134) (Reports, etc.)
Pinto, A. R., 1004 (Hel.)
——, with Ferreira, 998, 1202 (Ys.)
Pinto, Cesar & de Almeida, A. F., 901 (B.R.)
Pinto, D. B. & Penido, H. M., 765 (Hel.)
——, with —— & de Souza, 600 (Mal.)
Pinto, M. R., with Fonseca, de Azevedo, Amaro & Lacerda, 532 (Typh.)
——, with ——, —— & Lacerda, 532 (Typh.)
——, with ——, Gander, de Azevedo & Lacerda, 532 (Typh.)
——, with ——, Nunes & Lacerda, 532 (Typh.)
——, with ——, Oliveira, da Gama & Lacerda, 532 (Typh.)
van Pinxteren, J. A. C., with Hulshoff & Knoppers, 175 (B.R.)
Pipkin, A. C., with Azar & Garabedian, 141 (Hel.)
Pirastu, C., with Pansini, (1017) (Hel.)
Pires, W. da M., with de Bustamante, 897 (Ent.)
Piringer, W. A. & Hell, H. M., 979 (Rab.)
Piscaglia, A., 1058 (Mal.)
Pizzi, T., Agosin, M., Christen, R., Hoecker, G. & Neghme, A., 455 (Tryp.)
——, with Jarpa, Christen & Agosin, 456 (Tryp.)
Pizzi P., T. & Céspedes F., R., 467 (Am.)
Pla, J. C. & Muñoz Monteavaro, C., 359 (Am.)
Plooij, M., 857 (Typh.)
Ployé, M., 591 (Mal.)
Pluchon, J. & Pille, G., 468 (Am.)
Pochon, J., with P'An & Tchan, 847 (Pl.)
Poindexter, H. A., 52, 1211 (Hel.)
Ponce de Leon, E., with Ruiz Sanchez & Rueneling, 457 (Typh.)
Pons, C. A. & Reyes, F. M., 759 (Hel.)
Pool, C. L., 115 (Mal.)
Porte, with Nodenot, 242 (Am.)
Porte, L., 993 (Am.)
——, with Soulage, 539, (993) (Am.)
Potenza, L., 765 (Hel.)
——, Febres Cordero, R. & Anduze, P. J., 770 (Hel.)
——, with Luis González, Volgelsang, Anduze & Torres, 645 (Hel.)
——, with Vogelsang, 770 (Hel.)
Poul, J., (530) (Leish.)
Powell, H. M. & Culbertson, C. G., 733 (Rac.)
Pozsonyi, J., with Veghelyi, Kemeny & Sos, 772 (Def. Dis.)
Pozzo, A. A., 841 (Y.F.)
de Prada, J., 460 (Typh.)
——, Gay, B. & Llorente, A., 533 (Typh.)
——, with Parker, Bell & Lackman, 460 (Typh.)
Prasad, B. G., 219 (Leish.)
Prasad, G., with Seal, 356 (Pl.)
Pratesi, P., Raffa, L., Babudieri, B. & Nascimbene, A., 1091 (R.F.)
Pratt, C. K., with Hernández Morales, Oliver González & Maldonado, 764 (Hel.)
Pratt, H. O., with Stage, 1066 (Mal.)
Pratt, J. J., Jr., Heterick, R. H., Harrison, J. B. & Haber, L., 408 (Ent.)
Pratt-Thomas, H. R. & Switzer, P. K., 488 (Haem.)
Pražmowski, W., 126 (Typh.)
Prchal, C. J., with Sussman, (766) (Hel.)
Prejean, B. M., with Johansen, Ericsson, Wolcott, Meyer, Gray & Ross, 550 (Lep.)
Presnall, C. C., (735) (Rab.)
Prieto Lorenzo, A., (1209) (Hel.)
——, with Aparicio Garrido, 767 (Hel.)
Prior, I. A. M., with Brinkman, 1121 (Def. Dis.)
Prior, J. A., Cole, C. R. & Terbet, V., 399 (Der.)
di Prisco, J., with Mayer & di Prisco, 634 (Hel.)
di Prisco, R., with Mayer & di Prisco, 634 (Hel.)
Prommel, R., with Lagrange & Scheepmans, 633 (Hel.)
Proust, M., 491 (Haem.)
Pryer, H. B., with Freeman, (1229) (Prot.)
Public Health Rep., 152 (Pl.)
Puech, J., with Callot & Vermeil, 648 (Typh.)
Pugliese, J., with Benhamou, Albou & Destaing, 850 (Leish.)
Puig Solanes, M., 483 (Hel.)
Pujatti, D., 398 (Der.)
Puntigam, F., with Kaiser, 842 (Rac.)
Punzalare, N. M., with Rhende, Banner, Figueredo & Desai, 337 (Leish.)
Puyuelo, R., 464 (Hel.)

Q

Quarterman, K. D., with Mathis, 442 (Mal.)
——, with ——, Ferguson & Upnott, 842 (Ent.)
Quintanar, E. & Osorio, M. T., 263 (Hel.)
Quintos, F. N., with Stransky, 57 (Hel.)
Quiroga, J., 112 (Mal.)
Quiroga, M. I., Curia, L. & Ursi, C. G., (1457) (Leish.)

R

van Raalte, H. G. S., 358 (Am.)
Rabelo, E., 176 (B.R.)
Rabinowicz, K., with Yoeli, Steinitz & Gerichter, 37 (Am.)
Rabinowitz, K., with Silberstein & Gerichter, 234 (Rab.)
Rachou, R. G. & Azambuja, C. E. A., 590 (Mal.)
——, de Bustamante, F. M. & Ferreira, M. O., 600 (Mal.)
—— & Ferreira, M. O., 815 (Mal.)
——, with ——, 212 (Mal.)
——, Lima, M. M. & Lima, A. B., 805 (Mal.)
——, Neto, J. A. F. & Martins, C. M., 197 (Mal.)
——, Ricciardi, I., Luz, E. & Junior, J. G., 588 (Mal.)
Radice, J., with Negroni, 1124 (Der.)
Radosavljević, A., 1170 *bis* (Mal.)
Raffa, L., with Pratesi, Babudieri & Nascimbene, 1091 (R.F.)
Raffaele, G. & Coluzzi, A., 105 (Mal.)
Raffi, A., with Thiodet, Boulard & Fourrier, 111 (Mal.)
Ragab, H. A. A., 109, 213 (Mal.)
Rageau, J., with Grenier, 669 (Ent.)
——, with Roubaud & Colas-Belcour, 606 (Tryp.)
——, with Toumanoff, 104 (Mal.)
Raghavan, N. G. S. & Krishnan, K. S., 1021, 1110 (Hel.)
—— & Misra, B. G., 1023 (Hel.)
Rahm, U., with Geigy, 15 (Mal.)
Rai Chaudhuri, M. N., with Chaudhuri, 1175, 1176 (Mal.)
Raimondi, A. & Di Sapio, G., 556 (Hel.)
Rajamoney, P. D., with Reid & Chellappah, 714 (Mal.)
Raley, T. G., (715) (Mal.)
—— & Davis, E. D., 95 (Ent.)
——, with Rees & Davis, 163 (Ent.)
Ramachandra Rao, T., with Godbole & Viswanathan, (950) (Mal.)
——, with Viswanathan, 1062 (Mal.)
——, with —— & Juneja, 1063 (Mal.)
Ramakrishna, V., with Ramakrishnan & Krishnan, 441 (Mal.)
Ramakrishnan, S. P., Krishnan, K. S. & Ramakrishna, V., 441 (Mal.)
Ramamurthy, B., with Raman & Pinakapani, 643 (Hel.)
Raman, T. K., Ramamurthy, B. & Pinakapani, S., 643 (Hel.)
Ramirez-Rodriguez, E., with Rodriguez-Molina, Acevedo, Torres, Lopez-Sanabria & Diaz-Calderin, 1206 (Hel.)
Ramírez Villamediana, J. J. & Vergani, F., (1106) (Hel.)
Ramos, A. da S., with Coda, 430 (Mal.)
Ramos, I., with Torrealba & Riccardi, (725) (Tryp.)
Ranganathan, S. & Patwardhan, V. N., 266 (Haem.)
Ranganathan, S. K., Koshi, T. & Sitaraman, N. L., 415 (Ent.)
Ranque, J., Ranque, M. & Cabassu, H., 341 (Leish.)
——, with Sautet, (22) (Tryp.)
Ranque, M., with Ranque & Cabassu, 341 (Leish.)
Ransmeier, J. C., 87 (Misc. Dis.)
Ransome, G. A., 317 (Mal.)
Rao, B. A., 429 (Mal.)
Rao, G. S., 976 (Typh.)
Rao, K. N. A. & Kalra, S. L., 857 (R.F.)
——, with ——, 833, 835 (Typh.)
Rao, P. K., 1121 (Def. Dis.)
Rao, R. S. & Dogra, J. R., 33 (Rab.)
Raoult A., with Bergeret, 46 (R. F.)
Raper, A. B., 526 (Tryp.), 655 (Haem.)
——, with Lehmann, 267 (Haem.)
Raun, E. S., with Deonier, Peek, Davis & Notting ham, 443 (Mal.)
——, with Fluno, Deonier & Faulkner, 568 (Ent
Ravina, A., Guérin, P., Pécher, Y. & Clavel, B 989 (Am.)
——, Nory, J. & Avril, J., 404 (Misc. Dis.)
——, Pécher, Y. & Avril, J., 855 (R.F.)
Ray, A. P., with Singh & Nair, 1181 (Mal.)
——, with ——, —— & Basu, 1186 (Mal.)
Ray, H. N., (864) (Hel.)
Ray, J. C., with Ghosh & Ghosh, 829 (Leish.)
Read, C. P., (555) (Hel.)
Rees, B. E., Raley, T. G. & Davis, E. D., 163 (Ent
Rees, C. W., 981 (Am.)
——, with Phillips, 741 (Am.)
Rees-Wright, W., 501 (Lab.)
Reichenow, E., 116 (Mal.)
——, with Mudrow, 819 (Mal.)
Reid, C. C. & Flecker, H., 564 (Vms.)
Reid, J. A., with Rajamoney, P. D. & Chellappal L. R., 714 (Mal.)
——, with Wharton, 321 (Mal.)
——, with Wilson, 192 (Mal.)
Reilly, E. B. & Steel, H. H., 270 (Der.)
Reilly, J., Chen, G. & Geiling, E. M. K., 326, 71 (Mal.)
Reilly, W. A. & Earle, A. M., 1191 (Typh.)
Reinertson, J. W., with Thompson, Dunn Bayles, 741 (Am.)
Reiss, F., (224) (Typh.)
Relwicz, A. L., 860 (Lep.)
Remington, C. L., 1030 (Vms.)
Remlinger, P., (535), 734 (Rab.)
—— & Bailly, J., 535, (621), (1193), 1194 (Rab
Rempel, J. G., 1230 (Ent.)
Renifo, S., with Rey, 1170 (Mal.)
Renjifo-Salcedo, S., 720 (Tryp.)
—— & Orduz-Duarte, A., (1022) (Hel.)
—— & Osorno-Mesa, E., (970) (Tryp.)
Renton, H., with Charlewood & Shippel, 54 (Hel
Repass, R. P., with Bellamy, 953 (Mal.)
Resemann, G., with Burlingame, Gardne Cranmer & Vollmer, 358 (Am.)
Reul, R. & Eeraerts, W., 31 (Den.)
Rey, H. & Renifo, S., 1170 (Mal.)
—— & de Zulueta, J., 714 (Mal.)
Rey Vila, F., 958 (Mal.)
Reyes, F. M., with Pons, 759 (Hel.)
Reynes, V., 846 (Pl.)
——, with Alain, 1081 (Pl.)
Rheinboldt, H., Berti, F. A., Perego, C., Riec mann, B. H. G., Rzeppa, H. W., Mauri, A. C Hadler, W. A. & de Souza Lima, L., 548 (Lep
Rhodesia, Southern, 96, (791), (792) *bis*, (Report etc.)
Ribbands, C. R., 108, 196 (Mal.)
Ribeiro, D. O., with de Almeida, Lacaz & Azevedo, 775 (Der.)

Ribeiro, L. A., with Alves, (1019) (Hel.)
Ribo, with Botreau-Roussel & Sua, (998) (Ys.)
——, with Galliard, (1040) (Ent.)
Riccardi, B., with Torrealba & Ramos, (725) (Tryp.)
Ricciardi, I., (782) (Ent.)
——, with Rachou, Luz & Junior, 588 (Mal.)
Rice, C. E., with Frazier, 1122 (Haem.)
Richeson, E. M., with Greenberg, 1186 (Mal.)
Rickard, E. R., (457) (Typh.)
Ricketts, W. E., 228 *bis* (Bart.)
Riebeling, R., with Ruiz Sanchez & Ponce de Leon, 457 (Typh.)
Rieckmann, B., with Berti, Perego & Rzeppa, 372 (Lep.)
Rieckmann, B. H. G., with Rheinboldt, Berti, Perego, Rzeppa, Mauri, Hadler & de Souza Lima, 548 (Lep.)
Riedel, B. B., 558 (Hel.)
van Riel, J., 1133 (Reports, etc.)
Rigdon, R. H., (269) (Haem.)
—— & Breslin, D., 602 (Mal.)
—— & Ruskin, A., 14 (Mal.)
Rijkebüsch, L., with Bras, Kotter & Djoa Liang Ham, 83 (Der.)
Rissel, E. & Hartmann, G., 863 (Hel.)
Rivoalen, A. & Pham-Khac-Quang, (975) (Typh.) (988) (Am.)
Rivron, J., with Bertrand-Fontaine & Schneider, 532 (Typh.)
Robbins, M. L., Bourke, A. R. & Smith, P. K., 973 (Typh.)
Roberts, J. I., 397 (Haem.), 736, 1082 (Pl.)
Roberts, M., 1226 (Misc. Dis.)
Robertson, K. E. R., with Henderson & van Someren, 977 (Typh.)
Robertson, M., 707 (Mal.)
Robin, C. & Issa, D., 205 (Mal.)
—— & Serre, E., 1058 (Mal.)
Robin, L. A., (970), (975) *ter.* (Typh.)
Robinson, G. G., 586 (Mal.)
Robinson, J. H., with Travis, Morton & Jones, (114) (Mal.)
Robinson, L. B., with Ecker & Ambury, 124 (Leish.)
Robinson, W. A., with Galliard & Mille, 146 (Hel.)
Robinson, W. H., with Galliard & Mille, 60 (Hel.)
Roch, E., with Ortiz Mariotte, Varela & Gonzalez Robles, 973 (Typh.)
Rocha, A., with de Magalhães, 346 (Typh.)
——, with —— & Freire, 839 (Typh.)
Roche, P. J. L., 783 (Ent.)
Rockefeller Foundation, 165 (Reports, etc.)
de Rodaniche, E. C., 227 (Typh.)
Rodhain, J., 61 (Hel.), 523 (Mal.), 1219 (Der.), 1226, 1230 (Prot.)
—— & Hendrix, H., 89 (Prot.)
Rodhain, M. J., 601 (Mal.)
Rodier, J., with Méchali & Mallet, 310 (Mal.)
Rodrigues, P. M., with Travassos & Carrijo, 128 (Typh.)
Rodriquez-Molina, R., Acevedo, C. E., Torres, J. M., Lopez-Sanabria, U. & Ramirez-Rodriguez, E., with Diaz-Calderin, R., 1206 (Hel.)
Rogers, L., 461 (Chl.), 577 (B.R.)
Rogowsky, M., (666) (Misc. Dis.)
Rollo, I. M., Williamson, J. & Lourie, E. M., 333 (Tryp.)
Romaña, C. & Abalos, J. W., (530) (Leish.)
——, Nájera, L., Conejos, M. & Abalos, J. W., 125 (Leish.)
—— & Wygodzinsky, P., 1112 (Hel.)
Romeo Orbegozo, J. M., with Jiménez Díaz, Marina Fiol & Pérez Gòmez, 879 (Sp.)
Romeo Viamonte, J. M., with Clavero, (803), 812 (Mal.)
Römer, M., with Pifano, (656) *bis*
——, with ——, Mayer, Domínguez, Medina & Benaím Pinto, 723 (Tryp.)
Romero, A., Ibarra, A. B. & Fallas, M., 371 (Lep.)
de Rook, H., 206 (Mal.)
Roonwal, M. L., 28 (Typh.)
van Rooyen, C. E. & Scott, G. D., 221 (Typh.)
Rose, F. L., with Birtwell & Curd, (205) (Mal.)
——, with Curd, Davey & Hendry, 1185 (Mal.)
Rose, H. M., 349 (Typh.)
Rosebery, H., (207) (Mal.)
Rosemberg, J., Souza Campos, N. & Aun, J. N., 1095 (Lep.)
Rosenquist, R., with Obermayer & Bonar, 1098 (Lep.)
Rosenthal, N., with Murray, Baehr, Shwartzman, Mandelbaum, Doane, Weiss, Cohen & Snyder, 729 (Typh.)
Rosenthal, S. R. & Elmore, F. H., 776, 881 (Der.)
Ross, H., 471 (Lep.)
—— & Gemar, F., 1000 (Lep.)
——, with Johansen & Erickson, 547 (Lep.)
——, with ——, ——, Wolcott, Meyer, Gray & Prejean, 550 (Lep.)
Ross Institute Industrial Advisory Committee, 201 (Mal.)
Ross, M., with Dickson, 37 (Am.)
Ross, O. A. & Bueding, E., 633 (Hel.)
Ross, R. W., with Smithburn, Goodner, Dick & Kitchen, 229 (Y.F.)
Rossi-Espagnet, A., 641 (Hel.)
Rotberg, A. & Bechelli, L. M., 250 (Lep.)
Roth, A. R., with Lindquist & Yates, 892 (Ent.)
Roth, H., (392) (Hel.)
Rotter, H., (106) (Mal.)
Roubaud, E., Colas-Belcour, J. & Rageau, J. 606 (Tryp.)
Roy, A. T., with Muir, (1001) (Lep.)
Roy, I. B., 972 (Typh.)
Royo Montañes, M., (38) (Am.)
Ruhe, D. S., with Coatney, Cooper & Young, 318 (Mal.)
——, with ——, ——, —— & Burgess, 1171 (Mal.)
——, with Cooper & Coatney, 314 (Mal.)
Ruiz Reyes, F., 1117 (Hel.)
——, with Vargas, 557 (Hel.)
Ruiz-Sanchez, A., with Knight, Ruiz-Sanchez & McDermott, 458 (Typh.)
Ruiz Sanchez, F., 972 (Typh.)
——, with Knight, Ruiz-Sanchez & McDermott, 458 (Typh.)
——, Ponce de Leon, E. & Riebeling, R., 457 (Typh.)
Ruskin, A., with Rigdon, 14 (Mal.)

Russell, P. F., 809 (Mal.)
Ruysenaars, A., 850 (Am.)
Ryrie, G. A., 369, 1101 (Lep.)
Rzeppa, H. W., with Berti, Rieckmann & Perego, 372 (Lep.)
——, with Rheinboldt, Berti, Perego, Rieckmann, Mauri, Hadler & de Souza Lima, 548 (Lep.)

S

Sabrosky, C. W., 196 (Mal.)
Saccà, G., 669 (Ent.)
Sadun, E. H., 558 (Hel.)
——, Carrera, G. M., Krupp, I. M. & Allain, D. S., 852 (Am.)
Sadusk, J. F., Jr., 125 (Typh.)
Sáenz Vera, C., (132) (Pl.)
Salah, M. & Ghanem, M.D., 87 (Misc. Dis.)
Salcedo, J., Jr., Bamba, M.D., Carrasco, E. O., Jose, F. R. & Valenzuela, R. C., 560 (Def. Dis.)
——, Pedroche, A., Panganiban, E. C. & DeLeon, J. F., 73 (Def. Dis.)
Sanchez, G., with Engler, 484 (Hel.)
Sánchez Beltran, O., with Basnuevo, Guerra Valdés & Gutiérrez Estarli, (1200) (Am.)
Sandler, R., 39 (Am.)
Sangiorgi, G., 780 (Prot.)
San Julian, J., with Arana Iñiguez, Malosetti & Talice, 555 (Hel.)
Santiago Stevenson, D., with Hernández Morales, Oliver González & Maldonado, 477 (Hel.)
—— with ——, Pérez Santiago, Oliver González & Maldonado, 761 (Hel.)
Santos, J. P. (464) (Am.)
Santos, M., with Genú & de Oliveira Lima, 551 (Lep.)
Di Sapio, G., with Raimondi, 556 (Hel.)
Sasa, M., (307) (Mal.)
Sass, S., with Goldenson, Norton & Novak, 164 (Ent.)
Sasse, B. E. & Hackett, L. W., 1055 (Mal.)
Sauer, A., with Eichholtz, Hotovy & Weissflug, 1025 (Hel.)
Sautet, J., 556 (Hel.)
——, Asseo, S. & Cami, R., 36 (Am.)
—— & Ranque, J., (22) (Tryp.)
Sawitz, W. G., 1235 (B.R.)
Saxe, L. H., with Wenrich, (889) (Prot.)
Scaffidi, V. & Cali', G., 128 (Typh.)
Scaillet, L. & Haddad, E., 453 (Tryp.)
Scarlini, F., 743 (Am.)
Scharyj, M., with Campins & Gluck, 775 (Der.)
Scheecqmans, G., with Lagrange, 1105 (Hel.)
—— with —— & Promel, 633 (Hel.)
Scheid, G., with Mendheim, 68, 69, 1002 (Hel.)
—— & Mendheim, H., with Amenda, R., 1208 (Hel.)
Schein, M. W., 622 (Pl.)
Schiavi, A., with Wasicky & Unti, 276 (Ent.)
Schlesinger, R. W., 619 (Den.)
Schlieper, C. & Kalies, W., 556, 868 (Hel.)
Schlosser, R. J., 386 (Hel.)
Schmidt, H., 830 (Leish.), 870 (Hel.)
von Schmidt, H., 1235 (B.R.)
Schmidt, L. H., with Hughes, 965 (Mal.)
Schmitz, W. R., with Stierli, 569 (Ent.)
Schneider, C. R., with Schneider, 1182 (Mal.)
Schneider, H. H., 486 (Hel.)
Schneider, J., 758 (Lep.), 1055 (Mal.), 1114 (Hel.)
——, with Basset & Balle, 549 (Lep.)
——, with Bertrand-Fontaine, Fauvert & Hartmann, 433 (Mal.)
——, with —— & Rivron, 532 (Typh.)
——, with ——, Wolfromm & Cagnard, 67 (Hel.)
——, Decourt, P. & Montézin, G., 325 (Mal.)
——, with Durand & Dupoux, 957 (Mal.)
——, with Gaud & Méchali, 1176 (Mal.)
—— & Hartman, L., 311 (Mal.)
——, with Laviron & Lauret, 549 (Lep.)
——, Marchalant, Lotte, Buisson, B. & Diallo, 449 (Tryp.)
—— & Méchali, D., 204 (Mal.)
—— & Montézin, G., 43 (Am.), 1066 (Mal.)
—— & Schneider, C. R., 1182 (Mal.)
Schneider, R. G., Levin, W. C. & Haggard, M. E., 77 (Haem.)
Schnitzer, R. J. & Kelly, D. R., 826 (Tryp.)
Schoenaers, F., (607), 969 (Tryp.)
Schoeps, J., 160 (Prot.)
Schreiber, F. G. & Schubert, M., 142, 375 (Hel.)
Schroeder, E. F., with Most, Kane, Lavietes, Behm, Blum, Katzin & Hayman, 1009 (Mal.)
Schroeder, W., 801 (Mal.)
Schubert, J. H., with Morlan & Hill, 458 (Typh.)
Schubert, M., with Schreiber, 142, 375 (Hel.)
Schüffner, W., 70, 263, 649 (Hel.)
—— & Swellengrebel, N. H., 876 (Hel.)
Schujman, S., 472, 1097 (Lep.)
Schwetz, J., 634 (Hel.)
——, Baumann, H. & Fort, M., 191 (Mal.)
Scott, G. D., with van Rooyen, 221 (Typh.)
Scott, J. G., 883 (Oph.)
Scott, T. F. McN., with Sigel, Henle & Janton, 1078 (Typh.)
Scotti, G. & Tripodi, P., (641) (Hel), 745 (Am.)
Scudder, H. I. & Tarzwell, C. M., 569 (Ent.)
Sculthorpe, H. H., with Maegraith & Jones, 954 (Mal.)
Seabra, P., 438 (Mal.)
Seal, S. C., 234 (Pl.)
—— & Prasad, G., 356 (Pl.)
Seaton, D. R. & Adams, A. R. D., 12 (Mal.)
Sebag, A., with Dana, Cohen & Borsoni, 1074 (Typh.)
See, J., with Hunter, Bennett, Fry & Greene, 380 (Hel.)
Séguy, E., (779) (Misc. Dis.)
Seiler, H. E. & Norval, J., 1107 (Hel.)
Selbie, F. R., 837 (Typh.)
Selzer, G., with Irving, 73 (Def. Dis.)
Sen, N., with Dharmendra, 368 (Lep.)
Sen, P., 517, 518 (Mal.)
Seneca, H. & Henderson, E., 357 (Chl.)
Senevet, G., Mandoul, R. & Jacquemin, P., (276) (Ent.)
Sen Gupta, P. C., 340 (Leish.)
——, with Chakravarty, Bose & De, 726 (Leish.)
Sen Gupta, S., with Chatterjee, (135) (Am.)
Senigaglia, A., 299 (Mal.)
Sepulcri, P., 320 (Mal.)
Sergent, Ed., 283 (Reports, etc.), 303, 948 (Mal.)
Serre, E., with Robin, 1058 (Mal.)
Servino, V., with Amalfitano, Benetazzo, Cifarelli, Gambini & Tarizzo, 123 (Leish.)
Sewell, P. & Hawking, F., 1023 (Hel.)
——, with —— & Thurston, 1019 (Hel.)

Seydian, B., with Baltazard, Bahmanyar, Habibi & Mofidi, 1091 (R.F.)
——, with ——, Mofidi, Bahmanyar & Habibi, 995 (R.F.)
Shafar, J., 510 (B.R.)
Shah, B. R., with Adhia, (563) (Haem.)
Shanmugaratnam, K., (40) (Am.)
Sharp, J., with Israëls, (773) (Sp.)
Sharp, L. K., with Linnell, (522) (Mal.)
Shearman, C. E., 891 (Ent.)
Sheedy, J. A., 238 (Am.)
Shelley, H. M., 171 (B.R.)
Sheppard, E. H., with Fay, 95 (Ent.)
Sherif, A., with Ibrahim & Sorour, 40 (Am.)
Sherman, A. J., with Dawson & LaBoccetta, 566 (Ent.)
Shimada, Y., with Itoh, Kumano & Masu, 728 (Typh.)
Shimojo, H., with Tamiya, Hazato, Yamamoto & Nishioka, 344 (Typh.)
Shippel, S., with Charlewood & Renton, 54 (Hel.)
Short, D. J. & Parkes, A. S., (165) (Lab.)
Short, D. S., 1126 (Oph.)
Shortt, H. E., 4, 707, (Mal.), 121 (Tryp.), 284 (Reports, etc.), 1214 (Hel.)
——, Fairley, N. H., Covell, G., Shute, P. G. & Garnham, P. C. C., 193 (Mal.)
Shute, P. G., 276 (Ent.), 298, 707, (Mal.)
——, with Shortt, Fairley, Covell & Garnham, 193 (Mal.)
Shwartzman, G., with Murray, Baehr, Mandelbaum, Rosenthal, Doane, Weiss, Cohen & Snyder, 729 (Typh.)
Sicé, A. & Binder, T., 805 (Mal.)
Siebert, G., with Lang & Lönnecke, (478) (Hel.)
Siegert, R., 345, 612 (Typh.)
——, Simrock, W. & Ströder, U., 1077 (Typh.)
Sigalas, R., Pautrizel, R. & Noguès, C., 357 (Am.)
Sigel, M. M., Scott, T. F. McN., Henle, W. & Janton, O. H., 1078 (Typh.)
Siguier, F., Lamy & Trélat, J., 247 (Ys.)
——, Piette, Coblentz, B., Lemenager & Trélat, 1173 (Mal.)
Silberstein, W., 233 (Rab.)
——, Gerichter, C. B. & Rabinowitz, K., 234 (Rab.)
Siler, J. F., 1137 (Reports, etc.)
d'Silva, C. B., with Brooks, Lahiri & Ahuja, (842) (Rab.)
Silva C., R., with Noé, (609) (Tryp.)
Silva, F. & Monteiro, A. B., (266) (Def. Dis.)
da Silva, J. R., 377 (Hel.)
—— & da Costa, P. D., 376 (Hel.)
de Silva, P. C. C., 239 (Am.)
Silva, R., with Neghme, 464, 1197 (Am.)
——, with —— & Donckaster, (1107) (Hel.)
——, with ——, Vilches & Alfaro, 722 (Tryp.)
Silvany, A., Jr., with Pessôa, 1085 (Am.)
Simmons, S. W., with Hayes, (1132) (Ent.)
Simons, R. D. G. P., 371 (Lep.), 399, 1219 (Der.)
Simonson, E. & Keys, A., 519 (Mal.)
Simpson, G. G., 791 (Reports, etc.)
Simpson, I. A. & Molesworth, B. D., 860 (Lep.)
Simpson, T. W., with Mackie & Tuttle, 740 (Am.)
Simrock, W., with Hauss, 348 (Typh.)
——, with Siegert & Ströder, 1077 (Typh.)
Singer, K., Motulsky, A. G. & Wile, S. A., (881) (Haem.)
Singer, S. J., with Pauling, Itano & Wells, (267) (Haem.)
Singh, B., 49 (Lep.)
Singh, B. N., 623 (Am.)
Singh, G., Malik, K. S., Lahiri, M. N. & Ahuja, M. L., (848) (Chl.)
Singh, J., 1176 (Mal.)
—— & David, A., 1169 (Mal.)
——, Ray, A. P. & Nair, C. P., 1181 (Mal.)
——, ——, —— & Basu, P. C., 1186 (Mal.)
Sirry, A., 477 (Hel.)
Sisson, R. B., with King, Gilchrist, Wootton, Donaldson, Macfarlane, Jope, O'Brien, Peterson & Strangeways, 152 (Haem.)
Sitaraman, N. L., with Ranganathan & Koshi, 415 (Ent.)
Sivalingam, V., 632 (Hel.)
Skaliy, P. & Hayes, W. J., Jr., 409 (Ent.)
Sloan, N. R., Chung-Hoon, E. K., Godfrey-Horan, M. E. & Hedgcock, G. H., 1098 (Lep.)
Sluiter, C. Ph., 675 (B.R.)
Slutzky, Z., 744 (Am.)
Smadel, J. E., 837 (Typh.)
——, Bailey, C. A. & Diercks, F. H., 616 (Typh.)
——, with Bennett & Gauld, 223 (Typh.)
——, with Crocker, Bennett, Jackson, Snyder, Gauld & Gordon, 617 (Typh.)
——, with Ley & Woodward, 836 (Typh.)
——, Traub, R., Frick, L. P., Diercks, F. H. & Bailey, C. A., 615 (Typh.)
——, Woodward, T. E., Ley, H. L., Jr. & Lewthwaite, R., 129 (Typh.)
Smale, L. E. & Birsner, J. W., 85 (Der.)
Smit, A. M., with Gispen & Westermann, (129) (Typh.)
Smith, C. N., with Applewhite, 1232 (Ent.)
——, with Cole, 783 (Ent.)
——, Cole, M. M., Jones, E. A. & Clark, J. C., 279 (Ent.)
—— & King, W. V., 670 (Ent.)
Smith, C. S., with Tomlinson & Fogg, 160 (Prot.)
Smith, D. T., 156 (Der.)
Smith, G. F. & Geib, A. F., 277 (Ent.)
Smith, H. F. & Dy, F. J., 211 (Mal.)
——, —— & Cabrera, D. J., 1173, 1174 (Mal.)
——, —— & Del Rosario, F. G., (1170) (Mal.) 1234 (Ent.)
Smith, H. M. & Taylor, E. H., (1030) (Vms.)
Smith, J. F., with Smithburn, Mahaffy, Haddow & Kitchen, (620) (Den.)
Smith, M., 50, 372, 860 (Lep.), 1145 (B.R.)
——, with Lowe, 474 (Lep.)
Smith, P. K., with Robbins & Bourke, 973 (Typh.)
Smith, S. L. H. & Clow, J. M., 135 (Am.)
Smithburn, K. C., (32) (Den.)
——, Goodner, K., Dick, G. W. A., Kitchen, S. F. & Ross, R. W., 229 (Y.F.)
——, Haddow, A. J. & Gillett, J. D., (32) (Den.)
——, —— & Lumsden, W. H. R., (32) (Den.)
——, Mahaffy, A. F., Haddow, A. J., Kitchen, S. F. & Smith, J. F., (620) (Den.)
Sneath, P. A. T., 167 (Reports, etc.)
Snell, F. M., 654 (Haem.)
Snow, W. E., 804 (Mal.)
Snyder, J. C., with Bovarnick & Miller, 832 (Typh.)
——, with Fuller & Murray, 127 (Typh.)
——, with Murray, Baehr, Shwartzman, Mandelbaum, Rosenthal, Doane, Weiss & Cohen, 729 (Typh.)

Snyder, M. J., with Crocker, Bennett, Jackson, Smadel, Gauld & Gordon, 617 (Typh.)
Soberon y Parra, G. & Peláez, D., 360 (Am.)
Sodeman, W. A., 981 *bis* (Am.)
Sofia, F., 383, 636, 768 (Hel.)
Sohier, R. & Peloux, Y., 1090 (Am.)
Sokhey, S. S. & Habbu, M. K., 1081 (Pl.)
Soletta, L., 6 (Mal.), 1210 (Hel.)
Soman, D. W., 730, 1190 (Typh.)
Somasundaram, P., 60 (Hel.)
van Someren, E. C. C., (277), (889) *bis* (Ent.)
van Someren, G. R. C., with Cameron, 764 (Hel.)
van Someren, R. K. A., with Henderson & Robertson, 977 (Typh.)
de Sommerville, E. T. W., (1091) (Am.)
Sonnenberg, B., with Mackie, 788 (Reports, etc.)
Sorg, G., with Fessler & Fischer, 1169 (Mal.)
Sorour, A., with Ibrahim & Sherif, 40 (Am.)
Sós, J., with Véghelyi, Kemény & Pozsonyi, 772 (Def. Dis.)
Soto, M. C., 248 (Lep.)
Sotolongo, F., 257 (Hel.)
Soulage, J. & Porte, L., 539, (993) (Am.)
Soule, M. F., with Hanks, 250 (Lep.)
South Africa, Union of, 281 (Reports, etc.)
Southam, C. M., Thomson, A. E. & Burchenal, J. H., 392 (Hel.)
de Souza, E. F., with Penido & Pinto, 600 (Mal.)
de Souza, P. R., with de Souza Lima, 373 (Lep.)
de Souza-Araujo, H. C., 139, (375), 1002, (Lep.)
Souza Campos, N., with Rosemberg & Aun, 1095 (Lep.)
de Souza Lima, L., 547, 1098 (Lep.)
——, with Rheinboldt, Berti, Perego, Rieckmann, Rzeppa, Mauri & Hadler, 548 (Lep.)
—— & de Souza, P. R., 373 (Lep.)
Speechly, C. G. J., with Kalra, 834 (Typh.)
Sperbeck, T. M., 715 (Mal.)
Spicknall, C. G., Terry, L. L. & Coatney, G. R., 318 (Mal.)
Spies, T. D., with Garcia Lopez, Milanes, Lopez Toca, Aramburu & Lopez, 396 (Sp.)
——, with Suarez, Hernandez-Morales, Perez & Benitez-Gautier, 397 (Sp.)
Spingarn, C. L., with Edelman, 56 (Hel.)
Sprent, J. F. A., 145, 1108 (Hel.)
Sprunt, D. H., with McVay & Laird, 746 (Am.)
Squire, F. A., 527 (Tryp.)
Squires, B. T., 1033 (Misc. Dis.)
Stack, T., with Swellengrebel, 211 (Mal.)
Stage, H. H., 715 (Mal.)
—— & Berti, A. L., (815) (Mal.)
—— & Pratt, H. O., 1066 (Mal.)
Stahler, N., with Terzian, 305 (Mal.)
Stahnke, H. L., 656 (Vms.)
Standen, O. D., 552, 634 (Hel.)
Starkoff, I., with Starkoff, 1043 (Ent.)
Starkoff, O. & Starkoff, I., 1043 (Ent.)
Starzyk, J., 222 (Typh.)
—— & Westrych, F., 1075 (Typh.)
Staub, H., 10 (Mal.)
Steedman, H. F., 415 (Lab.)
Steel, H. H., with Reilly, 270 (Der.)
van Steenis, P. B., 393 (Def. Dis.)
Stefanopoulo, G. J., 1023, 1024 (Hel.)
—— & Ovazza, M., 390 (Hel.)
Stein, T., with Looney, 492 (Der.)
Steinitz, H., 38 (Am.)
——, with Yoeli, Gerichter & Rabinowicz, 37 (Am.)
Stevenson, D. S., with Hernández Morales, 256 (Hel.)
Stewart, G. T., 466 (Am.)
Stewart, I. S., 385 (Hel.)
——, with Davies, (759) (Hel.)
Steyn, D. G., 774 (Ep. Dropsy)
Steyn, P. J., with Odendaal, 73 (Def. Dis.)
Stickney, J. M., with Havens, (977) (Typh.)
Stierli, H. & Schmitz, W. R., 569 (Ent.)
Stirewalt, M. A., with Jachowski, 638 (Hel.)
Stoker, M. G. P., with MacCallum & Marmion, 226 (Typh.)
Stokes, E. J., with Hawksley, 970 (Typh.)
Stokinger, H. E., with Carpenter, Suhrland & Ackerman, 52 (Lep.)
Stoll, B., 434 (Mal.)
Stoppelman, M. R. H. & Blaauw-van Dok, S., 781 (Prot.)
Storey, N. E., with Kermack, (522) (Mal.)
Strandtmann, R. W., 348 (Typh.)
Strangeways, D. H., with King, Gilchrist, Wootton, Donaldson, Sisson, Macfarlane, Jope, O'Brien & Peterson, 152 (Haem.)
Stransky, E., Dauis-Lawas, D. F. & Vicente, C., 1217 (Def. Dis.)
—— & Quintos, F. N., 57 (Hel.)
Strauss, E. & Sulkin, S. E., 29 (Typh.)
Strauss, H., 651 (Hel.)
Strauss, M. J., with Colbert & Green, 778 (Der.)
Stray, E., with Hunter, Abbott & Pan, 379 (Hel.)
Stringer, A., with Page & Blackith, 93 (Ent.)
Ströder, U., with Siegert & Simrock, 1077 (Typh.)
Stunkard, H. W., 382 (Hel.), 981 (Am.)
Stursberg, H., 263 (Hel.)
Stutz, L., 1018 (Hel.)
Sua, H. B., with Botreau-Roussel & Ribo, (998) (Ys.)
Suarez, R. M., Spies, T. D., Hernandez-Morales, F. & Perez, E., with Benitez-Gautier, C., 397 (Sp.)
Suárez Torres, G., 733 (Rab.)
Subramaniam, H. & Chetty, K. N., (950) (Mal.)
Subramaniam, R., 275 (Misc. Dis.)
Subramanian, R. & Dixit, D. T., 431 (Mal.)
Suhrland, L. G., with Carpenter, Stokinger & Ackerman, 52 (Lep.)
Sulkin, S. E., Lodowski, C. H. & Hartman, L. W., 888 (Prot.)
——, with Strauss, 29 (Typh.)
Sullivan, B. H., Jr., 563 (Haem.)
Sullivan, T. D., McGregor, T., Eads, R. B. & Davis, D. J., 455 (Tryp.)
Sullivan, W. N., with Deonier, Nottingham & Lindquist, 13 (Mal.)
——, with McBride & Fulton, 786 (Ent.)
Sulman, F. G., 233 (Rab.)
Summers, W. A., 407 (Prot.)
Sun, C. J., 220 (Leish.)
Sun, Y.-P., 784 (Ent.)
Sundararaman, S., (161) (Ent.)
—— & Peffly, R. L., 323 (Mal.)
Sussman, O., (733) (Rab.)
——, with Parker, 130 (Typh.)
—— & Prchal, C. J., (766) (Hel.)

Sutter, V. A., with Deane, 1058 (Mal.)
——, with ——, Lédo, Freire, Cotrim & de Andrade, 1065 (Mal.)
Swartzwelder, J. C., 1218 (Vms.)
—— & Muller, G. R., 749 (Am.)
Swellengrebel, N. H., 193, (207), 707, 1053 (Mal.)
—— & Ihle, J. E. W., 675 (B.R.)
—— & Lodens, J. G., 211 (Mal.)
——, with Schüffner, 876 (Hel.)
—— & Stack, T., 211 (Mal.)
——, with Wilson & Garnham, 677 (Mal.)
Switzer, P. K., 397 (Haem.)
——, with Pratt-Thomas, 488 (Haem.)
Symington, T., with Giles, 348 (Typh.)
Symmers, W. St. C. & Valteris, K., 1131 (Ent.)
Symul, F., 1119 (Def. Dis.)
Szymoński, K., 113 (Mal.)

T

Taktikos, G., 272 (Oph.)
Talbot, D. R., Elerding, A. C. & Westwater, J. O., 591 (Mal.)
Taliaferro, L. G., with Taliaferro, 601, 1184 (Mal.)
Taliaferro, W. H. & Taliaferro, L. G., 601, 1184 (Mal.)
Talice, R., with Arana Iñiguez, Malosetti & San Julian, 555 (Hel.)
Talice, R. V., 121, 122 (Tryp.), (1027) (Hel.)
—— & Gurri, J., 479 *bis*, (1107) (Hel.)
Tamargo, A., with Faiguenbaum & Agosin, (1106) (Hel.)
Tamiya, T., Hazato, H., Yamamoto, T., Shimojo, H. & Nishioka, K., 344 (Typh.)
Tang, C. C., 384 (Hel.)
Tanganyika Territory, 167 (Reports, etc.)
Tanguy, F., with Auffret, 651 (Def. Dis.)
Tarizzo, M., with Amalfitano, Benetazzo, Cifarelli, Gambini & Servino, 123 (Leish.)
Tarun, D. W., with Knight & Miller, 360 (Am.)
Tarzwell, C. M., 673 (Ent.)
——, with Scudder, 569 (Ent.)
Tasqué, M., 483 (Hel.)
Taur, S. S., with Ling, 381 (Hel.)
Taylor, E. H., (1030) (Vms.)
——, with Smith, (1030) (Vms.)
Taylor, H. L., with Henschel & Keys, 519 (Mal.)
——, with Keys, Wells, Hoffbauer & Henschel, 519 (Mal.)
Tchan, Y. T., with P'An & Pochon, 847 (Pl.)
Tebbutt, A. H., 655 (Haem.)
Teichmann, G. O., 49, 51 (Lep.)
Telles, W., 1224 (Misc. Dis.)
TenBroeck, C., 844 (Rab.)
Tendeiro, J., 719 (Tryp.), 1022 (Hel.)
Teresa Herrero, M., (643) (Hel.)
Terry, E. D., with Knowles, 858 (R.F.)
Terry, L. L., with Spicknall & Coatney, 318 (Mal.)
Terzian, L. A. & Stahler, N., 305 (Mal.)
Tew, R. P., with Busvine, 894 (Ent.)
——, with David, 91 (Ent.)
Thelen, H. M., with Lennette & Meiklejohn, 30 (Typh.)
Théodoridès, J., 896, (897) (Ent.)
——, with Harant, 896 (Ent.)
Thiede, W. H., with Lincicome & Carpenter, 987 (Am.)
van Thiel, P. H., 159, 782 (Prot.)
Thillet, C. J., with Maldonado & Acosta Matienzo, 553 (Hel.)
——, with ——, Hernández Morales & Vélez Herrera, 872 (Hel.)
Thiodet & Fabregoule, 204 (Mal.)
—— & Fourrier, 110 (Mal.)
Thiodet, J., Boulard, G., Fourrier, A. & Raffi, A., 111 (Mal.)
—— & Fourrier, A., 315, 316 (Mal.)
Thompson, A. H., 223 (Typh.)
Thompson, B., 1225 (Misc. Dis.)
Thompson, G. A., 804 (Mal.)
Thompson, P. E., Dunn, M. C., Bayles, A. & Reinertson, J. W., 741 (Am.)
——, —— & Winder, C. V., 750 (R.F.)
Thomson, A. E., with Southam & Burchenal, 392 (Hel.)
Thurston, J. P., 1183 (Mal.)
——, with Hawking & Sewell, 1019 (Hel.)
Tibbs, R. O., with Oliphant, 732 (Den.)
Ticho, A., with Dostrovsky, 374 (Lep.)
Timisescu, F., with Ciuca, Ballif, Chelarescu, Vasiliu-Munteanu & Trofim, M. V., 593 (Mal.)
Ting, H. C., with Feng Lin & Hwang, 144 (Hel.)
Tisseuil, J., 367, 472, 1096 (Lep.), 585 (Mal.)
Tobie, E. J., with von Brand, Kissling & Adams, 218 (Tryp.)
——, von Brand, T. & Mehlman, B., 604 (Tryp.)
Tobie, J. E., 464 (Am.)
Todd, J. W., 417 (Reports, etc.)
Tokura, N., Tsai, C. W., Lo, J. P. & Chen, C. Y., 604 (Mal.)
Tolentino, P., with Astaldi, 153 (Haem.)
Tomaszewski, L., 127 (Typh.)
Tomlinson, T. H., Jr., Fogg, V. H. & Smith, C. S., 160 (Prot.)
Toms, B. A., 567 (Ent.)
Tôn-Thàt-Tùng, (988) (Am.)
Tooth, G., 1222 (Misc. Dis.)
Torbet, V., with Prior & Cole, 399 (Der.)
Torrealba, J. F., with Ramos, I. & Riccardi, B., (725) (Tryp.)
——, with Varadi, F., (721) (Tryp.)
Torrella, E., with Contreras & Guillén, 369, 373 (Lep.)
Torres, A. L., 273 (Oph.), 434 (Mal.)
Torres, C. M. & Duarte, E., 336 (Tryp.)
Torres E., P., with Luis González, Potenza, Vogelsang & Anduze, 645 (Hel.)
Torres, J. M., 556 (Hel.)
——, with Rodriguez-Molina, Acevedo, Lopez-Sanabria, Ramirez-Rodriguez & Diaz-Calderin, 1206 (Hel.)
Torrey, J. C. & Kahn, M. C., 15 (Mal.)
Toulant, M., 1128 (Oph.)
Toulant, P., 1128 (Oph.)
Toumanoff, C., 668, 891, (1040), (1044) *bis* (Ent.)
—— & Rageau, J., 104 (Mal.)
Tourenc, R., 521 (Mal.)
Touzin, R., with Blanc, 1104 (Hel.)
Town, B. W., Wills, E. D., Wilson, E. J. & Wormall, A., 1069 (Tryp.)
Trager, W. & McGhee, R. B., 816 (Mal.)
——, with ——, 1067 (Mal.)

Traub, R., 348 (Typh.)
—— & Frick, L. P., 616 (Typh.)
——, —— & Diercks, F. H., 1077 (Typh.)
——, with Philip, 566 (Ent.)
——, with Smadel, Frick, Diercks & Bailey, 615 (Typh.)
Travassos, J., with Pereira, 1076 (Typh.)
——, Pereira, H. G. & Vasconcelos, J. V., 346 (Typh.)
——, Rodrigues, P. M. & Carrijo, L. N., 128 (Typh.)
——, with Vasconcelos & Pereira, 1075 (Typh.)
Travis, B. V., 278 (Ent.)
—— & Lindquist, A. W., 892 (Ent.)
——, Morton, F. A., Jones, H. A. & Robinson, J. H., (114) (Mal.)
Tredre, R. F., 167 (Reports, etc.)
Trélat, with Siguier, Piette, Coblentz & Lemenager, 1173 (Mal.)
Trélat, J., with Siguier & Lamy, 247 (Ys.)
Trembley, H. L., with Coatney, Greenberg & Cooper, 444 (Mal.)
——, with Greenberg & Coatney, 603 (Mal.)
Triana, J. G., 752 (Ys.)
Trinquier, E., with Cavaillon, Adam, Bernard, Camain & Jaujou, 1179 (Mal.)
——, with Ceccaldi & Arnoult, 822 (Tryp.)
——, with ——, —— & Menu, 824 (Tryp.)
——, with ——, Pellissier & Arnoult, 528 (Tryp.)
——, with Pellissier, 459 (Typh.)
——, with —— & Troquereau, 617 (Typh.)
Tripodi, P., 124 (Leish.)
—— with Scotti, (641) (Hel.), 745 (Am.)
Trofim, M. V., with Ciuca, Balliff, Chelarescu, Timisescu & Vasiliu-Munteanu, 593 (Mal.)
Troquereau, P., with Pellissier & Trinquier, 617 (Typh.)
Truong-van-que, (955) (Mal.)
Tsai, C. W., with Tokura, Lo & Chen, 604 (Mal.)
Tsopellas, V., 1222 (Oph.)
Tuck, I. M. & Whittaker, N., (773) (Sp.)
Tuckman, E., 456 (Leish.), 1135 (Reports, etc.)
Tulpule, P. G., with Menon & Patwardhan, 1120 (Def. Dis.)
Tuncman, Z. M., (979) *bis* (Rab.)
Turner, E. E., with Hall & Mahboob, (1090) (Am.)
Turner, W. M. L., with Farquhar, 89 (Prot.)
Tuttle, R. L., with Mackie & Simpson, 740 (Am.)

U

Uchida, S., (90) (Ent.)
Unsworth, K., (244) (Am.)
Unti, O., with Wasicky & Schiavi, 276 (Ent.)
Upholt, W. M., with Mathis, Ferguson & Quarterman, 892 (Ent.)
Uribe-Piedrahita, C., 720 (Tryp.)
Urrets Zavalia, A., 1000 (Lep.)
Ursi, C. G., with Quiroga & Curia, (457) (Leish.)

V

Vaccaro, U., 639, 865 (Hel.)
Vaisman, A., with Levaditi & Barrat, (632) (Lep.)
——, with ——, Chaigneau & Barrat, 1103 (Lep.)
Valcke, G., 989 (Am.)
Valdés Larralde, L., with Basnuevo, (853) (Am.)
Vale, S., 176 (B.R.)
Valenzuela, R. C., with Salcedo, Bamba, Carrasco & Jose, 560 (Def. Dis.)
Valeri, C. M., with Chini, (77) (Haem.)
Valero, A., (459) (Typh.)
Valteris, K., with Symmers, 1131 (Ent.)
Vanbreuseghem, R., 201 (Mal.), 873 (Hel.)
van den Berghe, L. & Chardome, M., 644 (Hel.)
—— & Vander Borght, H., 264 (Def. Dis.)
——, Vincke, I. & Chardome, M., 816 (Mal.)
van den Bulcke, M. A., with Vincke, 522, 523 *bis* (Mal.)
Vander Borght, H., with van den Berghe, 264 (Def. Dis.)
Vanderplank, F. L., 820 (Tryp.)
Varadi, F., with Torrealba, (721) (Tryp.)
Varela, G. & Olarte, J., 1220 (Der.)
——, with Ortiz Mariotte, 225 (Typh.)
——, with ——, Roch & Gonzales Robles, 973 (Typh.)
Vargas, L., 149 (Hel.), (161), (668) (Ent.), 307 (Mal.)
—— & Diaz Nájera, A., 669 (Ent.)
—— & Ruiz Reyes, F., 557 (Hel.)
Vargues, R., with Giroud, 612 (Typh.)
——, with —— & Ciaccio, 613 (Typh.)
——, with Martin & Montel, 974 (Typh.)
Varwig, H., 338 (Leish.)
Vasconcelos, J. V., Travassos, J. & Pereira, H. G., 1075 (Typh.)
——, with —— & ——, 346 (Typh.)
Vasiliu-Munteanu, F., with Ciuca, Ballif, Chelarescu, Timisescu & Trofim, 593 (Mal.)
Vaz, E., 35, (536) (Rab.)
Vázquez Rosales, G., with Basnuevo, (853), (1200) (Am.)
Vega, V. S., with Lacaz, Iaria, Ferreira & Martins, 494 (Der.)
Vegas, M., Convit, J. & Espin, J., 631 (Lep.)
Véghelyi, P. V., Kemény, T. T., Pozsonyi, J. & Sós, J., 772 (Def. Dis.)
Velat, C. A., Weinstein, P. P. & Otto, G. F., 625 (Am.)
Vélez Herrera, F., with Maldonado, 553 (Hel.)
——, with ——, Hernández Morales & Thillet, 872 (Hel.)
Venkat Rao, V., 950 (Mal.)
Vercruysse, R., with van Grembergen & van Damme, (1211) (Hel.)
Vergani, F., with Ramírez Villamediana, (1106) (Hel.)
Verhaeghe, 455 (Tryp.)
Verlinde, J. D., (406) (Misc. Dis.)
——, Makstenieks, O., Beekman, H. & Copper, A. C., 1228 (Prot.)
Vermeil, C., with Callot & Puech, 618 (Typh.)
Vernant, P., with Donzelot & Kaufmann, 971 (Typh.)
Verolini, F., 326 (Mal.)
Vervent, G., with Colas-Belcour, 361, 362 (R.F.)
Vetter, M. H., with Nicholson, 848 (Pl.)
Vialat, C., with Béquignon, 352 (Rab.)
Vicente, C., with Stransky & Dauis-Lawas, 1217 (Def. Dis.)
Vieira, L. G., with Gonçalves, 1122 (Vms.)
v. Vietinghoff-Scheel, O. F., 1202 (Am.)
Vilain, 454 (Tryp.)
Vilain, M., 584 (Mal.)
Vilches, R., with Neghme, Silva & Alfaro, 722 (Tryp.)
Villasana, L., with Beltrán, 443 (Mal.)

Vincent, M., 403 (Ulc.)
Vincke, I., with van den Berghe & Chardome, 816 (Mal.)
Vincke, I. H. & van den Bulcke, M. A., 522, 523 *bis* (Mal.)
Vinelli, H., with Mackinnon, 84, 882 (Der.)
——, with —— & Montemayor, 84 (Der.)
Viswanathan, D. K., 1062 (Mal.)
——, with Godbole & Ramachandra Rao, (950) (Mal.)
—— & Ramachandra Rao, T., 1062 (Mal.)
——, —— & Juneja, M. R., 1063 (Mal.)
Vogel, H., 554, 862 (Hel.)
—— & Minning, W., 635 (Hel.)
——, with ——, 863 (Hel.)
Vogelsang, E. G. & Espín, J., (771) (Hel.)
—— & Herrera, L. A., (766) (Hel.)
—— & Potenza, L., 770 (Hel.)
Volgelsang, E., with Luis González, Potenza, Anduze & Torres, 645 (Hel.)
Vollmer, E., with Burlingame, Gardner, Resemann & Cranmer, 358 (Am.)
Vonwiller, P., (571) (Lab.)
Vucetich, M. & Giacobbe, O., 150 (Hel.)

W

Waddell, M. B., 31 (Y.F.)
Wagner, O., 878 (Hel.)
Wagner, W. H., 719 (Tryp.)
Wahi, A. L., (1173) (Mal.)
Wahlgren, F., with Gard, Magnusson & Gille, 1039 (Prot.)
Wallace, R. B., 1059 (Mal.)
Wallace, R. E., with Hanks, 368 (Lep.)
Wallace, W., with Hewitt & White, 1198 (Am.)
Wallace, W. S., with Hewitt, White, Hewitt, Hardy & Anduze, 1021 (Hel.)
Wallerstein, R. S., 253 (Hel.)
Wallertshauser, M., 158 (Misc. Dis.)
Walls, L. P., with Brownlee, Goss, Goodwin & Woodbine, 1070 (Tryp.)
——, with Copp, (456) (Tryp.)
——, with Goodwin, Goss & Lock, 1071 (Tryp.)
Walters, J. H., 530 (Leish.)
Walton, G. A., 208 (Mal.), 856 (R.F.), 1043 (Ent.)
Wan, C., with Wu & Chian, 665 (Ulc.)
Wang, A., with Huang, 145 (Hel.)
Wanson, M., Borgers, G. & Pannier, L., 873 (Hel.)
—— & Camphyn, R., 537 (Pl.)
——, with Courtois, 634 (Hel.)
——, Courtois, L. & Lebied, B., 647 (Hel.)
—— & Peel, E., 65 (Hel.)
——, —— & Lebied, B., 585 (Mal).
Warnecke, W., 877 (Hel.)
War Office, (96) (Reports, etc.)
Warren, V. G., with Hunter, 1112 (Hel.)
Warshaw, L. J., 792 (B.R.)
Washburn, W. L., (753) (Lep.)
Wasicky, R., Unti, O. & Schiavi, A., 276 (Ent.)
Waterhouse, P. D., with Collier, 1196 (Chl.)
——, with —— & Hall, 236, 1196 (Chl.)
Waterlow, J., 773 (Def. Dis.)
Watson, J. M., Zahawi, S., Naji, A. F. & Murtadha, M., 1203 (Hel.)
Watson, R. B., Paul, J. H. & Liang, K. C., 806 (Mal.)
Watson, R. S., with Bibile, Cullumbine, Kirtisinghe & Wickramanayake, (77) (Haem.), (87) (Misc. Dis.)
——, with ——, —— & Wikramanayake, 168 (Reports, etc.)
Watts, N. P., 55 (Hel.)
—— & Boyd, G. A., 1105 (Hel.)
Weathersbee, A. A., with Frohne, Williams & Hart, 709 (Mal.)
Wei, W. P., with Giroud, 1190 (Typh.)
Wei, Y., 132 (Chl.)
Weinstein, P. P., with Velat & Otto, 625 (Am.)
Weise, H., 1203 (Hel.)
Weiser, J., with Nikolic, 1108 (Hel.)
Weiss, L. B., with Blumberg & Doane, (27) (Typh.)
——, with Murray, Baehr, Shwartzman, Mandelbaum, Rosenthal, Doane, Cohen & Snyder, 729 (Typh.)
Weissflug, I., with Eichholtz, Hotovy & Sauer, 1025 (Hel.)
Welbourn, H. F., 561 (Haem.)
Welch, A. D., with Peters & Higashi, 147 (Hel.)
Weller, T. H., with Augustine, 143 (Hel.)
Wells, I. C., with Pauling, Itano & Singer, (267) (Haem.)
Wells, S., with Keys, Hoffbauer, Taylor & Henschel, 519 (Mal.)
Welsh, H. H., with Jellison, Elson & Huebner 731 (Typh.)
Wenderoth, H., 71 (Hel.)
Wenrich, D. H. & Saxe, L. H., (889) (Prot.)
Wenyon, C. E. M., with Andrews & Maegraith, 198 (Mal.)
——, with Maegraith & Andrews, 197 (Mal.)
Werner, H., 720 (Tryp.)
Westermann, C. D., with Gispen & Smit, (129) (Typh.)
Westphal, A., 133, 238 (Am.), 1056 (Mal.), 1130 (Prot.)
——, with Mohr, 1228 (Prot.)
Westrych, F., with Starzyk, 1075 (Typh.)
Westwater, J. O., with Talbot & Elerding, 591 (Mal.)
Weyer, F., 619, 832, 1074 (Typh.)
Wharton, G. W., 224 (Typh.)
Wharton, R. H. & Reid, J. A., 321 (Mal.)
Whitcomb, F. C., with Kunstadter & Milzer, (83) (Der.)
White, E., with Hewitt, Hewitt, Hardy, Wallace & Anduze, 1021 (Hel.)
——, with —— & Wallace, 1198 (Am.)
White, F. B., 623 (Chl.)
White, W. C., with Coatney, Cooper, Culwell & Imboden, 1177, (Mal.)
——, with ——, ——, Lints, Culwell & Eyles, 1060 (Mal.)
——, with Lints, Coatney, Cooper, Culwell & Eyles, 1059 (Mal.)
Whitfield, G. H., with Haworth & McKenna, (360) (Am.)
Whitney, D. M., with Anigstein & Beninson, (29) (Typh.)
Whittaker, N., with Tuck, (773) (Sp.)
Whittick, J. W., 731 (Typh.)
Whittingham, H. & Galley, R. A. E., 93 (Ent.)
Wickham, N. & Carne, H. R., 668 (Prot.)
Wickramanayake, T. W., with Bibile, Cullumbine, Kirtisinghe & Watson, (77) (Haem.)

Wiesmann, R. & Lotmar, R., 278 (Ent.)
Wigand, R., 759 (Hel.)
Wigglesworth, A., 167, 787 (Reports, etc.), 201 (Mal.)
Wikramanayake, T., with Bibile, Cullumbine, Kirtisinghe & Watson, (87) (Misc. Dis.)
——, with Cullumbine & Bibile, (1216) (Def. Dis.)
Wikramanayake, T. W., with Bibile, Cullumbine & Watson, 168 (Reports, etc.)
Wiktor, T. J., 889 (Prot.)
Wilder, R. M., (612) (Typh.)
Wile, S. A., with Singer & Motulsky, (881) (Haem.)
Wilkinson, P. R., 388 (Hel.)
Williams, G. M., with Frohne, Weathersbee & Hart, 709 (Mal.)
Williamson, A. D. & Leong, P. C., 560 (Def. Dis.)
Williamson, J., 334 (Tryp.)
——, with Rollo & Lourie, 333 (Tryp.)
Wills, E. D., with Town, Wilson & Wormall, 1069 (Tryp.)
—— & Wormall, A., 1070 (Tryp.)
Wilmot, A. J., with Armstrong & Elsdon-Dew, 994 (Am.)
Wilson, D. B., 300, 516, 517 (Mal.)
——, Garnham, P. C. C. & Swellengrebel, N. H., 677 (Mal.)
Wilson, E. J., with Town, Wills & Wormall, 1069 (Tryp.)
—— & Wormall, A., 120 (Tryp.)
Wilson, T., 1213 (Hel.)
—— & Reid, J. A., 192 (Mal.)
Winckel, C. W. F., 317 (Mal.)
——, Kok, K., de Jongh, D. K. & Knoppers, A. T., 712 (Mal.)
Winder, C. V., with Thompson & Dunn, 750 (R.F.)
Windsor, E., (623) (Chl.)
Winer, L. H., 1030 (Der.)
Winsser, J. & Makstenieks, O., 781 *bis* (Prot.)
Winter, P. D., 475, 1102 (Lep.)
Wiradikarta, D. & Darwis, A., 49 (Lep.)
Wiśniowski, J., 844 (Rab.)
Wohlrab, R., 346 *bis* (Typh.)
Wolcott, R. R., with Johansen, Erickson, Meyer, Gray, Prejean & Ross, 550 (Lep.)
Wolff, W. A. & Harrell, G. T., 351 (Typh.)
Wolfromm, R., with Bertrand-Fontaine, Schneider & Cagnard, 67 (Hel.)
Wolfs, J. & Devignat, R., 637 (Hel.)
Wolstenholme, B., with Gear & Cort, 977 (Typh.)
Won, W. D., 1195 (Pl.)
Wong, S. C. & Cox, H. R., 29 (Typh.)
Woo, D., with Koo, 144 (Hel.)
Wood, E. C., with Jones, (165) (Lab.)
Woodbine, M., with Brownlee, Goss, Goodwin & Walls, 1070 (Tryp.)
Woodruff, A. W., with Murgatroyd, 262 (Hel.)
Woodruff, C. E., with Kelly, 659 (Der.)
Woodville, H. C., with Harrison, 1081 (Pl.)
Woodward, T. E., with Ley & Smadel, 836 (Typh.)
——, with Smadel, Ley & Lewthwaite, 129 (Typh.)
Woolf, C. R., 974 (Typh.)
Wootton, I. D. P., with King, Gilchrist, Donaldson, Sisson, Macfarlane, Jope, O'Brien, Peterson & Strangeways, 152 (Haem.)
Wormall, A., with Town, Wills & Wilson, 1069 (Tryp.)
——, with Wills, 1070 (Tryp.)
——, with Wilson, 120 (Tryp.)
Worms, R., 833 (Typh.)
——, Péquignot, H. & Held, J. P., 971 (Typh.)
Woyciechowska, S., with Kryński, 1074 (Typh.)
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